WEST Search History

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DATE: Thursday, September 02, 2004

Hide?	Set Name	Query	Hit Count
	DB=USPT,PGPB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L5	L4 and @pd > 20030717	13
☐	L4	L2 and cos$ library	68
☐	L3	L2 same cos$ library	0
☐	L2	L1 same yeast	1197
☐	L1	homologous recombination	19373

END OF SEARCH HISTORY

FILE 'EMBASE' ENTERED AT 14:38:10 ON 02 SEP 2004

FILE 'CAPLUS' ENTERED AT 14:38:10 ON 02 SEP 2004

=> s homologous recombination and yeast
L1 2137 HOMOLOGOUS RECOMBINATION AND YEAST

=> s l1 and review
L2 220 L1 AND REVIEW

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 151 DUP REM L2 (69 DUPLICATES REMOVED)

=> d bib abs 1-10

L3 ANSWER 1 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2004328767 EMBASE
TI The mechanism of vertebrate nonhomologous DNA end joining and its role in V(D)J recombination.
AU Lieber M.R.; Ma Y.; Pannicke U.; Schwarz K.
CS M.R. Lieber, USC Norris Comprehensive Cancer Ctr., Rm. 5428, Univ. S. California Keck Sch. M., Los Angeles, CA, United States. lieber@usc.edu
SO DNA Repair, (2004) 3/8-9 (817-826).
Refs: 81
ISSN: 1568-7864 CODEN: DRNEAR
PUI S 1568-7864(04)00074-6
CY Netherlands
DT Journal; General Review
FS 022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
AB The vertebrate immune system generates double-strand DNA (dsDNA) breaks to
generate the antigen receptor repertoire of lymphocytes. After those double-strand breaks have been created, the DNA joinings required to complete the process are carried out by the nonhomologous DNA end joining pathway, or NHEJ. The NHEJ pathway is present not only in lymphocytes, but in all eukaryotic cells ranging from ***yeast*** to humans. The NHEJ pathway is needed to repair these physiologic breaks, as well as challenging pathologic breaks that arise from ionizing radiation and oxidative damage to DNA. .COPYRGT. 2004 Elsevier B.V. All rights reserved.

L3 ANSWER 2 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 1
AN 2004327959 EMBASE
TI DSB repair: The ***yeast*** paradigm.
AU Aylon Y.; Kupiec M.
CS M. Kupiec, Dept. Molec. Microbiol. and Biotech., Tel Aviv University, Ramat Aviv, Israel. martin@post.tau.ac.il
SO DNA Repair, (2004) 3/8-9 (797-815).
Refs: 247
ISSN: 1568-7864 CODEN: DRNEAR
PUI S 1568-7864(04)00144-2
CY Netherlands
DT Journal; General Review
FS 004 Microbiology
022 Human Genetics
LA English
SL English
AB Genome stability is of primary importance for the survival and proper functioning of all organisms. Double-strand breaks (DSBs) arise spontaneously during growth, or can be created by external insults. In response to even a single DSB, organisms must trigger a series of events to promote repair of the DNA damage in order to survive and restore chromosomal integrity. In doing so, cells must regulate a fine balance between potentially competing DSB repair pathways. These are generally classified as either ***homologous*** ***recombination*** (HR) or non-homologous end joining (NHEJ). The ***yeast*** Saccharomyces cerevisiae is an ideal model organism for studying these repair processes. Indeed, much of what we know today on the mechanisms of repair in eukaryotes come from studies carried out in budding ***yeast*** . Many of the proteins involved in the various repair pathways have been isolated and the details of their mode of action are currently being unraveled at the molecular level. In this ***review*** , we focus on exciting new work eminating from ***yeast*** research that provides fresh insights into the DSB repair process. This recent work supplements and complements the wealth of classical genetic research that has been performed in ***yeast*** systems over the years. Given the conservation of the repair mechanisms and genes throughout evolution, these studies have profound implications for other eukaryotic organisms. .COPYRGT. 2004 Elsevier B.V. All rights reserved.

L3 ANSWER 3 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 2
AN 2004321071 EMBASE
TI Sequence-specific modification of mouse genomic DNA mediated by gene targeting techniques.
AU Sangiuolo F.; Novelli G.
CS Dr. G. Novelli, Dept. Biopathology Diagn. Imaging, Tor Vergata University, Via Montpellier 1, IT-00133 Rome, Italy. novelli@med.uniroma2.it
SO Cytogenetic and Genome Research, (2004) 105/2-4 (435-441).
Refs: 51
ISSN: 1424-8581 CODEN: CGRYAJ
CY Switzerland
DT Journal; General Review
FS 022 Human Genetics
030 Pharmacology
037 Drug Literature Index
039 Pharmacy
LA English
SL English
AB The major impact of the human genome sequence is the understanding of disease etiology with deduced therapy. The completion of this project has shifted the interest from the sequencing and identification of genes to the exploration of gene function, signalling the beginning of the post-genomic era. Contrasting with the spectacular progress in the identification of many morbid genes, today therapeutic progress is still lagging behind. The goal of all gene therapy protocols is to repair the precise genetic defect without additional modification of the genome. The main strategy has traditionally been focused on the introduction of an expression system designed to express a specific protein, defective in the transfected cell. But the numerous deficiencies associated with gene augmentation have resulted in the development of alternative approaches to treat inherited and acquired genetic disorders. Among these one is represented by gene repair based on ***homologous*** ***recombination*** (HR). Simply stated, the process involves targeting the mutation in situ for gene correction and for restoration of a normal gene function. ***Homologous*** ***recombination*** is an efficient means for genomic manipulation of prokaryotes, ***yeast*** and some lower eukaryotes. By contrast, in higher eukaryotes it is less efficient than in the prokaryotic system, with non- ***homologous*** ***recombination*** being 10-50 fold higher. However, recent advances in gene targeting and novel strategies have led to the suggestion that gene correction based on HR might be used as clinical therapy for genetic disease. This site-specific gene repair approach could represent an alternative gene therapy strategy in respect to those involving the use of retroviral or lentiviral vectors to introduce therapeutic genes and linked regulatory sequences into random sites within the target cell genome. In fact, gene therapy approaches involving addition of a gene by viral or nonviral vectors often give a short duration of gene expression and are difficult to target to specific populations of cells. The purpose of this paper is to ***review*** oligonucleotide-based gene targeting technologies and their applications on modifying the mouse genome. Copyright .COPYRGT. 2004 S. Karger AG, Basel.

L3 ANSWER 4 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 3
AN 2004165134 EMBASE
TI New insights into the mechanism of ***homologous*** ***recombination*** in ***yeast*** .
AU Aylon Y.; Kupiec M.
CS M. Kupiec, Dept. Molec. Microbiol. and Biotech., Tel Aviv University, Ramat Aviv 69978, Israel. martin@post.tau.ac.il
SO Mutation Research - Reviews in Mutation Research, (2004) 566/3 (231-248).
Refs: 150
ISSN: 1383-5742 CODEN: MRRRFK
PUI S 1383-5742(03)00114-5
CY Netherlands
DT Journal; General Review
FS 004 Microbiology
LA English
SL English
AB Genome stability is of primary importance for the survival and proper functioning of all organisms. Double-strand breaks (DSBs) arise spontaneously during growth, or can be created by external insults. Repair of DSBs by ***homologous*** ***recombination*** provides an efficient and fruitful pathway to restore chromosomal integrity. Exciting new work in ***yeast*** has lately provided insights into this complex process. Many of the proteins involved in recombination have been isolated and the details of the repair mechanism are now being unraveled at the molecular level. In this ***review*** , we focus on recent studies which dissect the recombinational repair of a single broken chromosome. After DSB formation, a decision is made regarding the mechanism of repair (recombination or non-homologous end-joining). This decision is under genetic control. Once committed to the recombination pathway, the broken chromosomal ends are resected by a still unclear mechanism in which the DNA damage checkpoint protein Rad24 participates. At this stage several proteins are recruited to the broken ends, including Rad51p, Rad52p, Rad55p, Rad57p, and possibly Rad54p. A genomic search for homology ensues,
followed by strand invasion, promoted by the Rad51 filament with the participation of Rad55p, Rad57p and Rad54p. DNA synthesis then takes place, restoring the resected ends. Crossing-over formation depends on the length of the homologous recombining sequences, and is usually counteracted by the activity of the mismatch repair system. Given the

conservation of the repair mechanisms and genes throughout evolution, these studies have profound implications for other eukaryotic organisms. .COPYRGT. 2003 Elsevier B.V. All rights reserved.

L3 ANSWER 5 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2004072353 EMBASE
TI [SPO11: An activity that promotes DNA breaks required for meiosis].
SPO11: UNE ACTIVITE DE COUPURE DE L'ADN INDISPENSABLE A LA MEIOSE.
AU Baudat F.; De Massy B.
CS B. De Massy, Institut de Genetique Humaine, CNRS UPR 1142, 141, rue de la Cardonille, 34396 Montpellier Cedex 05, France. bdemassy@igh.cnrs.fr
SO Medecine/Sciences, (2004) 20/2 (213-218).
Refs: 32
ISSN: 0767-0974 CODEN: MSMSE4
CY France
DT Journal; General Review
FS 002 Physiology
029 Clinical Biochemistry
LA French
SL English; French
AB Recombination between homologous chromosomes during meiosis is an essential process, which mechanistical function is to ensure the reductional segregation of chromosomes at the first meiotic division. SPO11, one of the key genes directly involved in this process, has been at the origin of considerable interest for the past five years, for several reasons. First, Spo11 is responsible for the initiation of meiotic recombination through the formation of DNA double-strand breaks by a type II DNA topoisomerase-like activity. Moreover, Spo11, and its function, have been conserved through evolution, from yeasts to human, as demonstrated by the identification of members of the Spo11 protein family and the analyses of corresponding mutants. Indeed, for every eukaryote that has been tested, spo11 mutants are deficient for meiotic recombination and are partially or completely sterile. Depending on the species, this reduced fertility reflects either a defect in chromosome segregation, or an arrest response in germ cell differentiation. Similarities and differences from species to species uncover a complex set of regulations that coordinate recombination with other events of meiotic prophase, such as chromosome pairing and meiotic cell cycle.

L3 ANSWER 6 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2004341992 EMBASE
TI Coupling ***homologous*** ***recombination*** with growth selection in ***yeast*** : A tool for construction of random DNA sequence libraries.
AU Schaerer-Brodbeck C.; Barberis A.
CS C. Schaerer-Brodbeck, ESBATech AG, Wagistrasse 21, CH-8952 Schlieren, Switzerland. schaerer@esbatech.com
SO BioTechniques, (2004) 37/2 (202-206).
Refs: 16
ISSN: 0736-6205 CODEN: BTNQDO
CY United States
DT Journal; General Review
FS 029 Clinical Biochemistry
LA English

L3 ANSWER 7 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 4
AN 2004178849 EMBASE
TI Genetic aspects of targeted insertion mutagenesis in yeasts.
AU Klinner U.; Schafer B.
CS U. Klinner, RWTH Aachen, Institut fur Biologie IV, Worringer Weg, D-52056 Aachen, Germany. ulrich.klinner@rwth-aachen.de
SO FEMS Microbiology Reviews, (2004) 28/2 (201-223).
Refs: 212
ISSN: 0168-6445 CODEN: FMREE4
PUI S 0168-6445(03)00091-3
CY Netherlands
DT Journal; General Review
FS 004 Microbiology
LA English
SL English
AB Targeted insertion mutagenesis is a main molecular tool of ***yeast*** science initially applied in Saccharomyces cerevisiae. The method was extended to fission ***yeast*** Schizosaccharomyces pombe and to "non-conventional" ***yeast*** species, which show specific properties of special interest to both basic and applied research. Consequently, the behaviour of such non-Saccharomyces yeasts is reviewed against the background of the knowledge of targeted insertion mutagenesis in S. cerevisiae. Data of homologous integration efficiencies obtained with circular, ends-in or ends-out vectors in several yeasts are compared. We follow details of targeted insertion mutagenesis in order to recognize possible rate-limiting steps. The route of the vector to the target and possible mechanisms of its integration into chromosomal genes are considered. Specific features of some ***yeast*** species are discussed. In addition, similar approaches based on ***homologous*** ***recombination*** that have been established for the mitochondrial genome of S. cerevisiae are described. .COPYRGT. 2003 Federation of European Microbiological Societies. Published by Elsevier B.V. All rights reserved.

L3 ANSWER 8 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 5
AN 2004220545 EMBASE
TI Strategies for gene disruptions and plasmid constructions in fission ***yeast*** .
AU Wang L.; Kao R.; Ivey F.D.; Hoffman C.S.
CS C.S. Hoffman, Boston College, Biology Department, Higgins Hall 401B, Chestnut Hill, MA 02467, United States. hoffmacs@bc.edu
SO Methods, (2004) 33/3 (199-205).
Refs: 22
ISSN: 1046-2023 CODEN: MTHDE
PUI S 1046-2023(03)00313-X
CY United States
DT Journal; General Review
FS 004 Microbiology
LA English
SL English
AB Molecular genetic analyses in Schizosaccharomyces pombe are greatly enhanced by our ability to delete chromosomal genes via ***homologous*** ***recombination*** and to introduce genes expressed from autonomous plasmids. In this paper, we describe a novel approach to generating marked deletion cassettes that bypasses the need for the long, PAGE-purified oligonucleotides required in the currently used PCR-based deletion approach. We also describe additional uses of this two-step PCR method for constructing chromosomal insertion cassettes. Finally, we describe how gap repair in S. pombe can facilitate plasmid constructions in a manner that circumvents the reliance on compatible restriction sites in the DNA molecules that are being joined. Several applications of this gap repair plasmid construction strategy are discussed. .COPYRGT. 2003 Elsevier Inc. All rights reserved.

L3 ANSWER 9 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 6
AN 2004072471 EMBASE
TI Post-replication repair in DT40 cells: Translesion polymerases versus recombinases.
AU Hochegger H.; Sonoda E.; Takeda S.
CS H. Hochegger, Department of Radiation Genetics, Faculty of Medicine, Kyoto University, Sakyo-ku, 606-8501 Kyoto, Japan
SO BioEssays, (2004) 26/2 (151-158).
Refs: 79
ISSN: 0265-9247 CODEN: BIOEEJ
CY United States
DT Journal; General Review
FS 004 Microbiology
022 Human Genetics
LA English
SL English
AB Replication forks inevitably stall at damaged DNA in every cell cycle. The ability to overcome DNA lesions is an essential feature of the replication machinery. A variety of specialized polymerases have recently been discovered, which enable cells to replicate past various forms of damage by a process termed translesion synthesis. Alternatively, ***homologous*** ***recombination*** can be used to restart DNA replication across the lesion. Genetic and biochemical studies have shed light on the impact of these two post-replication repair pathways in bacteria and ***yeast*** . In vertebrates, however, a genetic approach to study post-replication repair has been compromised because many of the genes involved appear to be essential for embryonic development. We have taken advantage of the chicken cell line DT40 to perform a genetic analysis of translesion synthesis and ***homologous*** ***recombination*** and to characterize genetic interactions between these two pathways in vertebrates. In this article, we aim to summarize our current understanding of post-replication repair in DT40 in the perspective of bacterial, ***yeast*** and mammalian genetics. .COPYRGT. 2004 Wiley Periodicals, Inc.

L3 ANSWER 10 OF 151 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN DUPLICATE 7
AN 2004:258850 BIOSIS
DN PREV200400259889
TI DNA double-strand break repair by ***homologous*** ***recombination*** .
AU Dudas, Andrej; Chovanec, Miroslav [Reprint Author]
CS Laboratory of Molecular Genetics, Cancer Research Institute, Slovak Academy of Sciences, Vlarska 7, 833 91, Bratislava, 37, Slovakia miroslav.chovanec@savba.sk
SO Mutation Research, (March 2004) Vol. 566, No. 2, pp. 131-167. print. ISSN: 0027-5107 (ISSN print).
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 19 May 2004
Last Updated on STN: 19 May 2004
AB DNA double-strand breaks (DSB) are presumed to be the most deleterious DNA
lesions as they disrupt both DNA strands. ***Homologous***

recombination (HR), single-strand annealing, and non-homologous end-joining are considered to be the pathways for repairing DSB. In this ***review*** , we focus on DSB repair by HR. The proteins involved in this process as well as the interactions among them are summarized and characterized. The main emphasis is on eukaryotic cells, particularly the budding ***yeast*** Saccharomyces cerevisiae and mammals. Only the RAD52 epistasis group proteins are included.

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FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 14:38:10 ON 02 SEP 2004
L1 2137 S HOMOLOGOUS RECOMBINATION AND YEAST
L2 220 S L1 AND REVIEW
L3 151 DUP REM L2 (69 DUPLICATES REMOVED)

=> d bib abs 11-50

L3 ANSWER 11 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN
AN 2004052674 EMBASE
TI The Mus81 solution to resolution: Generating meiotic crossovers without Holliday junctions.
AU Hollingsworth N.M.; Brill S.J.
CS S.J. Brill, Dept. of Molec. Biol. and Biochem., Rutgers University, Piscataway, NJ 08854, United States. brill@mbcl.rutgers.edu
SO Genes and Development, (15 Jan 2004) 18/2 (117-125).
Refs: 61
ISSN: 0890-9369 CODEN: GEDEEP
CY United States
DT Journal; General Review
FS 022 Human Genetics
029 Clinical Biochemistry
LA English

L3 ANSWER 12 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2004:204859 CAPLUS
DN 140:351192
TI Repairing a double-strand chromosome break by ***homologous*** ***recombination*** : revisiting Robin Holliday's model
AU Haber, James E.; Ira, Gregorz; Malkova, Anna; Sugawara, Neal
CS Rosenstiel Center and Department of Biology, Brandeis University, Waltham, MA, 02454-9110, USA
SO Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences (2004), 359(1441), 79-86
CODEN: PTRBAE; ISSN: 0962-8436
PB Royal Society
DT Journal; General Review
LA English
AB A ***review*** , with refs. Since the pioneering model for ***homologous*** ***recombination*** proposed by Robin Holliday in 1964, there has been great progress in understanding how recombination occurs at a mol. level. In the budding ***yeast*** Saccharomyces cerevisiae, one can follow recombination by phys. monitoring DNA after the synchronous induction of a double-strand break (DSB) in both wild-type and mutant cells. A particularly well-studied system has been the switching of ***yeast*** mating-type (MAT) genes, where a DSB can be induced synchronously by expression of the site-specific HO endonuclease. Similar studies can be performed in meiotic cells, where DSBs are created by the Spo11 nuclease. There appear to be at least two competing mechanisms of ***homologous*** ***recombination*** : a synthesis-dependent strand annealing pathway leading to noncrossovers and a two-end strand invasion mechanism leading to formation and resoln. of Holliday junctions (HJs), leading to crossovers. The establishment of a modified replication fork during DSB repair links gene conversion to another important repair process, break-induced replication. Despite recent revelations, almost 40 yr after Holliday's model was published, the essential ideas he proposed of strand invasion and heteroduplex DNA formation, the formation and resoln. of HJs, and mismatch repair, remain the basis of our thinking.
RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 13 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN DUPLICATE 8
AN 2004076062 EMBASE
TI Protein localization in proteomics.
AU Davis T.N.
CS T.N. Davis, Department of Biochemistry, University of Washington, Box 357350, Seattle, WA 98195-7350, United States. tdavis@u.washington.edu
SO Current Opinion in Chemical Biology, (2004) 8/1 (49-53).
Refs: 29
ISSN: 1367-5931 CODEN: COCBF4
CY United Kingdom
DT Journal; General Review
FS 029 Clinical Biochemistry
LA English
SL English
AB A global analysis of the localization of 4156 ***yeast*** proteins has just been accomplished. Smaller scale analyses have been performed in a variety of organisms. These studies typically use green fluorescent protein as a tag for proteins in living cells. Improvements in the yellow and sapphire color variants will increase their utility. Reengineering of the red fluorescent protein has produced faster maturing tetrameric and monomeric variants not prone to aggregation. Techniques for high-throughput tagging of proteins include integration by ***homologous*** ***recombination*** , integration using mobile elements or recombinational cloning to produce plasmids expressing fusion proteins. Alternatives to localizing tagged proteins are to use antibodies or aptamers to detect the untagged protein.

L3 ANSWER 14 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:530782 CAPLUS
DN 139:209361
TI A new link for a linker histone
AU Conconi, Antonio; Wellinger, Raymund J.
CS Department de Microbiologie et Infectiologie, Universite de Sherbrooke, Sherbrooke, QC, J1H 5N4, Can.
SO Molecular Cell (2003), 11(6), 1421-1423
CODEN: MOCEFL; ISSN: 1097-2765
PB Cell Press
DT Journal; General Review
LA English
AB A ***review*** with refs. and commentary on the research of Downs et al. (ibid. 2003, 11, 1685-1692). Classically, the functions of linker histones (histones H1 and variants) have been related mainly to chromatin organization and the ensuing consequences on transcription. Remarkably, ***yeast*** histone H1 may not comply, as it appears to regulate ***homologous*** ***recombination*** specifically.
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 15 OF 151 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN DUPLICATE 9
AN 2003:424293 BIOSIS
DN PREV200300424293
TI New 'marker swap' plasmids for converting selectable markers on budding ***yeast*** gene disruptions and plasmids.
AU Voth, Warren P.; Jiang, Yi Wei; Stillman, David J. [Reprint Author]
CS Department of Pathology, University of Utah Health Sciences Center, Salt Lake City, UT, 84132, USA
david.stillman@path.utah.edu
SO Yeast, (August 2003) Vol. 20, No. 11, pp. 985-993. print.
ISSN: 0749-503X (ISSN print).
DT Article
LA English
ED Entered STN: 17 Sep 2003
Last Updated on STN: 17 Sep 2003
AB Marker swap plasmids can be used to change markers for genes disrupted with nutritional markers in the ***yeast*** Saccharomyces cerevisiae. We describe 18 new marker swap plasmids, and we also ***review*** other plasmids available for marker conversions. All of these plasmids have long regions of flanking sequence identity, and thus the efficiency of ***homologous*** ***recombination*** mediated by marker conversion is very high. Marker swaps allow one to easily perform crosses to construct double mutant strains even if each of the disrupted strains contains the same marker, as is the case with the KanMX marker used in the ***yeast*** knockout collection. Marker swaps can also be used to change the selectable marker on plasmids, eliminating the need for subcloning.

L3 ANSWER 16 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2004:115745 CAPLUS
DN 140:352114
TI Non-homologous DNA end joining
AU Pastwa, Elzbieta; Blasiak, Janusz
CS Department of Medicinal Chemistry, Medical University of Lodz, Lodz, Pol.
SO Acta Biochimica Polonica (2003), 50(4), 891-908
CODEN: ABPLAF; ISSN: 0001-527X
PB Polish Biochemical Society
DT Journal; General Review
LA English
AB A ***review*** . DNA double-strand breaks (DSBs) are a serious threat for the cell and when not repaired or misrepaired can result in mutations or chromosome rearrangements and eventually in cell death. Therefore, cells have evolved a no. of pathways to deal with DSB including ***homologous*** ***recombination*** (HR), single-strand annealing (SSA) and non-homologous end joining (NHEJ). In mammals DSBs are primarily repaired by NHEJ and HR, while HR repair dominates in ***yeast*** , but this depends also on the phase of the cell cycle. NHEJ functions in all kinds of cells, from bacteria to man, and depends on the structure of DSB termini. In this process two DNA ends are joined directly, usually with no sequence homol., although in the case of same polarity of the single stranded overhangs in DSBs, regions of microhomol. are utilized. The usage of microhomol. is common in DNA end-joining of physiol. DSBs, such as at the coding ends in V(D)J (variable(diversity) joining) recombination. The main components of the NHEJ system in eukaryotes are the catalytic subunit of DNA protein kinase (DNA-PKcs), which is recruited by DNA Ku protein, a heterodimer of Ku70 and Ku80, as well as XRCC4 protein and DNA ligase IV. A complex of Rad50/Mre11/Xrs2, a

family of Sir proteins and probably other yet unidentified proteins can be also involved in this process. NHEJ and HR may play overlapping roles in the repair of DSBs produced in the S phase of the cell cycle or at replication forks. Aside from DNA repair, NHEJ may play a role in many different processes, including. The maintenance of telomeres and integration of HIV-1 genome into a host genome, as well as the insertion of pseudogenes and repetitive sequences into the genome of mammalian cells. Inhibition of NHEJ can be exploited in cancer therapy in radio-sensitizing cancer cells. Identification of all key players and fundamental mechanisms underlying NHEJ still requires further research.
RE.CNT 128 THERE ARE 128 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 17 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN
AN 2003087552 EMBASE
TI Genetic requirements for the targeted integration of Agrobacterium T-DNA in Saccharomyces cerevisiae.
AU van Attikum H.; Hooykaas P.J.J.
CS P.J.J. Hooykaas, Inst. of Molecular Plant Sciences, Leiden University, Clusius Laboratory, Wassenaarseweg 64, 2333 AL Leiden, Netherlands. hooykaas@rulbim.leidenuniv.nl
SO Nucleic Acids Research, (1 Feb 2003) 31/3 (826-832).
Refs: 35
ISSN: 0305-1048 CODEN: NARHAD
CY United Kingdom
DT Journal; General Review
FS 004 Microbiology
LA English
SL English
AB Agrobacterium tumefaciens delivers transferred DNA (T-DNA) into cells of plants and ***yeast*** . In plants, the T-DNA integrates at random positions into the genome by non- ***homologous*** ***recombination*** (NHR), whereas in ***yeast*** the T-DNA preferably integrates by ***homologous*** ***recombination*** (HR). Here we show that T-DNA integration by HR in ***yeast*** requires the recombination/repair proteins Rad51 and Rad52, but not Rad50, Mre11, Xrs2, Yku70 and Lig4. In the HR events a remarkable shift from insertion-type events to replacement events was observed in rad50, mre11 and xrs2 mutants. Residual integration in the rad51 mutant occurred predominantly by HR, whereas in the rad52 mutant integration occurred exclusively by NHR. Previously, we found that T-DNA integration by NHR is abolished in a yku70 mutant. Thus, Rad52 and Yku70 are the key regulators of T-DNA integration, channeling integration into either the HR or NHR pathway.

L3 ANSWER 18 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
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AN 2003461946 EMBASE
TI Dna Mismatch Repair: Molecular Mechanisms and Biological Function.
AU Schofield M.J.; Hsieh P.
CS M.J. Schofield, Genetics and Biochemistry Branch, Natl. Inst. Diabet., Digest. K., National Institutes of Health, Bethesda, MD 20892, United States. schofiel@helix.nih.gov
SO Annual Review of Microbiology, (2003) 57/- (579-608).
Refs: 181
ISSN: 0066-4227 CODEN: ARMIAZ
CY United States
DT Journal; General Review
FS 004 Microbiology
016 Cancer
037 Drug Literature Index
048 Gastroenterology
LA English
SL English
AB DNA mismatch repair (MMR) guards the integrity of the genome in virtually all cells. It contributes about 1000-fold to the overall fidelity of replication and targets mispaired bases that arise through replication errors, during ***homologous*** ***recombination*** , and as a result of DNA damage. Cells deficient in MMR have a mutator phenotype in which the rate of spontaneous mutation is greatly elevated, and they frequently exhibit microsatellite instability at mono- and dinucleotide repeats. The importance of MMR in mutation avoidance is highlighted by the finding that defects in MMR predispose individuals to hereditary nonpolyposis colorectal cancer. In addition to its role in postreplication repair, the MMR machinery serves to police ***homologous*** ***recombination*** events and acts as a barrier to genetic exchange between species.

L3 ANSWER 19 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN
AN 2004129165 EMBASE
TI Holliday junctions in the eukaryotic nucleus: Resolution in sight?.
AU Heyer W.-D.; Ehmsen K.T.; Solinger J.A.
CS W.-D. Heyer, Division of Biological Sciences, Section of Microbiology, University of California, Davis, CA 95616-8665, United States. wdheyer@ucdavis.edu
SO Trends in Biochemical Sciences, (2003) 28/10 (548-557).
Refs: 68
ISSN: 0968-0004 CODEN: TBSCDB
PUI S 0968-0004(03)00220-2
CY United Kingdom
DT Journal; General Review
FS 004 Microbiology
LA English
SL English
AB The Holliday junction is a key recombination intermediate whose resolution generates crossovers. Interplay between recombination, repair and replication has moved the Holliday junction to the center stage of nuclear DNA metabolism. Holliday junction resolvases in the eukaryotic nucleus have long eluded identification. The endonucleases Mus81/Mms4-Eme1 and XPF-MEI-9/MUS312 are structurally related to the archaeal resolvase Hjc and were found to be involved in crossover formation in budding ***yeast*** and flies, respectively. Although these endonucleases might represent one class of eukaryotic resolvases, their substrate preference opens up the possibility that junctions other than classical Holliday junctions might contribute to crossovers. Holliday junction resolution to non-crossover products can also be achieved topologically, for example, by the action of RecQ-like DNA helicases combined with topoisomerase III.

L3 ANSWER 20 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN
AN 2003168471 EMBASE
TI The Escherichia coli RecA protein complements recombination defective phenotype of the Saccharomyces cerevisiae rad52 mutant cells.
AU Dudas A.; Markova E.; Vlasakova D.; Kolman A.; Batosova Z.; Brozmanova J.; Chovanec M.
CS J. Brozmanova, Department of Molecular Genetics, Cancer Research Institute, Slovak Academy of Sciences, Vlarska 7, 833 91 Bratislava 37, Slovakia. jela.brozmanova@savba.sk
SO Yeast, (15 Apr 2003) 20/5 (389-396).
Refs: 69
ISSN: 0749-503X CODEN: YESTE3
CY United Kingdom
DT Journal; General Review
FS 004 Microbiology
LA English
SL English
AB The Saccharomyces cerevisiae rad52 mutants are sensitive to many DNA damaging agents, mainly to those that induce DNA double-strand breaks (DSBs). In the ***yeast*** , DSBs are repaired primarily by ***homologous*** ***recombination*** (HR). Since almost all HR events are significantly reduced in the rad52 mutant cells, the Rad52 protein is believed to be a key component of HR in S. cerevisiae. Similarly to the S. cerevisiae Rad52 protein, RecA is the main HR protein in Escherichia coli. To address the question of whether the E. coli RecA protein can rescue HR defective phenotype of the rad52 mutants of S. cerevisiae, the recA gene was introduced into the wild-type and rad52 mutant cells. Cell survival and DSBs induction and repair were studied in the RecA-expressing wild-type and rad52 mutant cells after exposure to ionizing radiation (IR) and methyl methanesulphonate (MMS). Here, we show that expression of the E. coli RecA protein partially complemented sensitivity and fully complemented DSB repair defect of the rad52 mutant cells after exposure to IR and MMS. We suggest that in the absence of Rad52, when all endogenous HR mechanisms are knocked out in S. cerevisiae, the heterologous E. coli RecA protein itself presumably takes over the broken DNA. Copyright .COPYRGT. 2003 John Wiley & Sons, Ltd.

L3 ANSWER 21 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN DUPLICATE 10
AN 2003481833 EMBASE
TI RecQ helicases and topoisomerase III in cancer and aging.
AU Laursen L.V.; Bjergbaek L.; Murray J.M.; Andersen A.H.
CS A.H. Andersen, Department of Molecular Biology, Aarhus University, C.F. Mollers Alle, Bldg. 130, 8000 Aarhus-C, Denmark. aha@mb.au.dk
SO Biogerontology, (2003) 4/5 (275-287).
Refs: 102
ISSN: 1389-5729 CODEN: BIOGCN
CY Netherlands
DT Journal; General Review
FS 005 General Pathology and Pathological Anatomy
016 Cancer
021 Developmental Biology and Teratology
022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
AB RecQ helicases have in recent years attracted increasing attention due to the important roles they play in maintaining genomic integrity, which is essential for the life of a cell and the survival of a species. Humans with mutations in RecQ homologues are cancer prone and suffer from premature aging. A great effort has therefore been made to understand the molecular mechanisms and the biological pathways, in which RecQ helicases are involved. It has become clear that these enzymes work in close concert with DNA topoisomerase III, and studies in both ***yeast*** and mammalian systems point to a role of the proteins in processes involving ***homologous*** ***recombination*** . In this ***review*** we discuss the genetic and biochemical evidence for possible functions of RecQ helicases and DNA topoisomerase III in multiple cellular processes such as DNA recombination, DNA replication, and cell cycle checkpoint

control.

L3 ANSWER 22 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:91992 CAPLUS
DN 138:349084
TI Sister chromatid cohesion and genome stability in vertebrate cells
AU Morrison, C.; Vagnarelli, P.; Sonoda, E.; Takeda, S.; Earnshaw, W. C.
CS Institute of Cell and Molecular Biology, Wellcome Centre for Cell Biology, University of Edinburgh, Edinburgh, UK
SO Biochemical Society Transactions (2003), 31(1), 263-265
CODEN: BCSTB5; ISSN: 0300-5127
PB Portland Press Ltd.
DT Journal; General Review
LA English
AB A ***review*** . For successful eukaryotic mitosis, sister chromatid pairs remain linked after replication until their kinetochores have been attached to opposite spindle poles by microtubules. This linkage is broken at the metaphase-anaphase transition and the sisters sep. In budding ***yeast*** , this sister chromatid cohesion requires a multi-protein complex called cohesin. A key component of cohesin is Scc1/Mcd1 (Rad21 in fission ***yeast***). Disruption of the chicken orthologue of Scc1 by gene targeting in DT40 cells causes premature sister chromatid sepn. Cohesion between sister chromatids is likely to provide a substrate for post-replicative DNA repair by ***homologous*** ***recombination*** . In keeping with this role of cohesion, Scc1 mutants also show defects in the repair of spontaneous and induced DNA damage. Scc1-deficient cells frequently fail to complete metaphase chromosome alignment and show chromosome segregation defects, suggesting aberrant kinetochore function. Consistent with this, the chromosomal passenger protein, INCENP (inner centromere protein) fails to localize to centromeres. Survivin, another passenger protein and one which interacts with INCENP, also fails to localize to centromeres in Scc1-deficient cells. These results show that cohesin maintains genomic stability by ensuring appropriate DNA repair and equal chromosome segregation at mitosis.
RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 23 OF 151 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 11
AN 2003:189825 BIOSIS
DN PREV200300189825
TI Mitotic recombination in Saccharomyces cerevisiae.
AU Prado, Felix; Cortes-Ledesma, Felipe; Huertas, Pablo; Aguilera, Andres [Reprint Author]
CS Departamento de Genetica, Facultad de Biologia, Universidad de Sevilla, 41012, Sevilla, Spain
aguilo@us.es
SO Current Genetics, (January 2003) Vol. 42, No. 4, pp. 185-198. print.
ISSN: 0172-8083 (ISSN print).
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 16 Apr 2003
Last Updated on STN: 16 Apr 2003
AB Mitotic ***homologous*** ***recombination*** (HR) is an important mechanism for the repair of double-strand breaks and errors occurring during DNA replication. It is likely that the recombinational repair of DNA lesions occurs preferentially by sister chromatid exchanges that have no genetic consequences. However, most genetically detectable HR events occur between homologous DNA sequences located at allelic positions in homologous chromosomes, or between DNA repeats located at ectopic positions in either the same, homologous or heterologous chromosomes. Mitotic recombination may occur by multiple mechanisms, including double-strand break repair, synthesis-dependent strand annealing, break-induced replication and single-strand annealing. The occurrence of one recombination mechanism versus another depends on different elements, including the position of the homologous partner, the initiation event, the length of homology of the recombinant molecules and the genotype. The genetics and molecular biology of the ***yeast*** Saccharomyces cerevisiae have proved essential for the understanding of mitotic recombination mechanisms in eukaryotes. Here, we ***review*** recent genetic ***yeast*** data that contribute to our understanding of the different mechanisms of mitotic recombination and the in vivo role of the recombination proteins.

L3 ANSWER 24 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN
AN 2003142425 EMBASE
TI Temporal and spatial parameters of skeletal gene expression: Targeting RUNX factors and their coregulatory proteins to subnuclear domains.
AU Stein G.S.; Lian J.B.; Stein J.L.; van Wijnen A.J.; Choi J.-Y.; Pratap J.; Zaidi S.K.
CS Dr. G.S. Stein, Department of Cell Biology, Univ. of Massachusetts Cancer Center, 55 Lake Ave. North, Worcester, MA 01655, United States.
gary.stein@umassmed.edu
SO Connective Tissue Research, (2003) 44/SUPPL. 1 (149-153).
Refs: 44
ISSN: 0300-8207 CODEN: CVTRBC
CY United Kingdom
DT Journal; General Review
FS 001 Anatomy, Anthropology, Embryology and Histology
021 Developmental Biology and Teratology
022 Human Genetics
LA English
SL English
AB Key components of the basal transcription machinery and several tissue-specific transcription factor complexes are functionally compartmentalized as specialized subnuclear domains. We have identified a unique 31-38 amino acid targeting signal (NMTS) that directs the Runx (Cbfa/AML) transcription factors to distinct nuclear matrix-(NM) associated sites within the nucleus that support gene expression. Our determination of the NMTS crystal structure, ***yeast*** 2 hybrid screens to identify NM interacting proteins, and in situ colocalization studies with Runx interacting factors (YAP, Smad, TLE) suggest that localization of Runx transcription factors at intranuclear sites facilitates the assembly and activity of regulatory complexes that mediate activation and suppression of target genes. Mice homozygous for the deletion of the intranuclear Runx2 targeting signal in a ***homologous*** ***recombination*** (Runx2 .DELTA.C) do not form bone due to maturational arrest of osteoblasts, demonstrating the importance of fidelity of subnuclear localization for tissue-differentiating activity. These results provide evidence that Runx2 subnuclear targeting and the associated regulatory functions are essential for a spatiotemporal placement that facilitates activation of Runx-dependent genes involved in tissue differentiation during embryonic development.

L3 ANSWER 25 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN
AN 2002384539 EMBASE
TI Generation of disruption cassettes in vivo using a PCR product and Saccharomyces cerevisiae.
AU Zaragoza O.
CS O. Zaragoza, Department of Microbiology, Albert Einstein College of Medicine, Golding Building, 1300 Morris Park Avenue, Bronx, NY 10461, United States. ozaragoz@aecom.yu.edu
SO Journal of Microbiological Methods, (1 Jan 2003) 52/1 (141-145).
Refs: 16
ISSN: 0167-7012 CODEN: JMIMDQ
PUI S 0167-7012(02)00154-9
CY Netherlands
DT Journal; General Review
FS 004 Microbiology
LA English
SL English
AB A method to obtain disruption cassettes based on the ***homologous*** ***recombination*** in Saccharomyces cerevisiae is described. The disruption marker is amplified by PCR using oligonucleotides containing 50 bp homologous to the disruptable gene and 20 bp from the marker. The PCR product is cotransformed into ***yeast*** with a plasmid containing the gene. After recombination, a plasmid that carries the disruption cassette for the gene is produced. .COPYRGT. 2003 Elsevier Science B.V. All rights reserved.

L3 ANSWER 26 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN DUPLICATE 12
AN 2003495574 EMBASE
TI Regulation of alternative replication bypass pathways at stalled replication forks and its effects on genome stability: A ***yeast*** model.
AU Barbour L.; Xiao W.
CS W. Xiao, Dept. of Microbiology and Immunology, University of Saskatchewan, 107 Wiggins Road, Saskatoon, Sask. S7N 5E5, Canada. wei.xiao@usask.ca
SO Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis,
(27 Nov 2003) 532/1-2 (137-155).
Refs: 167
ISSN: 0027-5107 CODEN: MRFMEC
CY Netherlands
DT Journal; General Review
FS 004 Microbiology
LA English
SL English
AB Replication-blocking lesions result in increased genomic instability by stalling replication forks. Eukaryotic cells appear to have evolved several surveillance and repair/bypass mechanisms to ensure that replication can be resumed at these stalled forks. In the ***yeast*** Saccharomyces cerevisiae, the helicases Srs2 and Sgs1 appear to play a role in controlling the processing and stabilization of stalled replication forks. These proteins appear to be tightly regulated throughout the cell cycle and play a direct role in DNA-damage checkpoints. This allows the cells to determine the best mechanism to reestablish replication at the stalled fork: by shuttling the lesion into the RAD6-dependent pathway that can lead to error-free or error-prone bypass; or by using ***homologous*** ***recombination*** . Under conditions where both the RAD6-dependent pathway and recombination are disabled, the cells can bypass the lesion using a novel damage avoidance mechanism that is controlled by Mgs1. Replication fork bypass processes appear to be highly conserved within eukaryotes, with homologs for SGS1 and MGS1 found in both Schizosaccharomyces pombe and mammalian cells.

L3 ANSWER 27 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:923873 CAPLUS
DN 140:158376
TI Role of the error-free damage bypass postreplication repair pathway in the maintenance of genomic stability
AU Smirnova, Marina; Klein, Hannah L.
CS Kaplan Comprehensive Cancer Center, Department of Biochemistry, New York
University School of Medicine, New York, NY, 10016, USA
SO Mutation Research (2003), 532(1,2), 117-135
CODEN: MUREAV; ISSN: 0027-5107
PB Elsevier Science B.V.
DT Journal; General Review
LA English
AB The postreplication repair pathway (PRR) is composed of error-free and error-prone sub-pathways that allow bypass of DNA damage-induced replication-blocking lesions. The error-free sub-pathway is also used for bypass of spontaneous DNA damage and functions in cooperation with recombination pathways. In diploid ***yeast*** cells, error-free PRR is needed to prevent genomic instability, which is manifest as loss of heterozygosity (LOH) events of increased chromosome loss and recombination. ***Homologous*** ***recombination*** acts synergistically with the error-free damage avoidance branch of PRR to prevent chromosome loss. The DNA damage checkpoint gene MEC1 acts synergistically with the PRR pathway in maintaining genomic stability. Integration of the PRR pathway with other cellular pathways for preventing genomic instability is discussed. In diploid strains, the most dramatic increase is in the abnormality of chromosome loss when a repair or damage detection pathway is defective. These data are preceded by a ***review*** of the post replication repair pathway in Saccharomyces cerevisiae, with emphasis on the error-free damage avoidance sub-pathway.
RE.CNT 82 THERE ARE 82 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 28 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN DUPLICATE 13
AN 2003492869 EMBASE
TI ***Homologous*** ***Recombination*** and Gene Targeting in Plant Cells.
AU Reiss B.
CS B. Reiss, Max-Planck-Inst. Zuechtungsforsch., Carl-von-Linne-Weg 10, D-50829 Koln, Germany
SO International Review of Cytology, (2003) 228/- (85-139).
Refs: 274
ISSN: 0074-7696 CODEN: IRCYAJ
CY United States
DT Journal; General Review
FS 004 Microbiology
LA English
SL English
AB Gene targeting has become an indispensable tool for functional genomics in ***yeast*** and mouse; however, this tool is still missing in plants. This ***review*** discusses the gene targeting problem in plants in the context of general knowledge on recombination and gene targeting. An overview on the history of gene targeting is followed by a general introduction to genetic recombination of bacteria, ***yeast*** , and vertebrates. This abridged discussion serves as a guide to the following sections, which cover plant-specific aspects of recombination assay systems, the mechanism of recombination, plant recombination genes, the relationship of recombination to the environment, approaches to stimulate ***homologous*** ***recombination*** and gene targeting, and a description of two plant systems, the moss Physcomitrella patens and the chloroplast, that naturally have high efficiencies of gene targeting. The ***review*** concludes with a discussion of alternatives to gene targeting.

L3 ANSWER 29 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:477088 CAPLUS
DN 139:112230
TI Transposable elements as tools for genomics and genetics in Drosophila
AU Ryder, Edward; Russell, Steven
CS Department of Genetics, University of Cambridge, Cambridge, CB2 3EH, UK
SO Briefings in Functional Genomics & Proteomics (2003), 2(1), 57-71
CODEN: BFGPAL; ISSN: 1473-9550
PB Henry Stewart Publications
DT Journal; General Review
LA English
AB A ***review*** . The P-element has been the workhorse of Drosophila genetics since it was developed as a tool for transgenesis in 1982; the subsequent development of a variety of systems based on the transposon have provided a range of powerful and flexible tools for genetics and genomics applications. P-element insertions are frequently used as starting-points for generating chromosomal deletions to remove flanking genes, either by screening for imprecise excision events or by selecting for male recombination events. Elements that utilize the ***yeast*** FLP/FLP recombination target (FRT) site-specific recombination system have been widely used to generate molecularly marked mitotic clones for mosaic anal., extending the reach of this powerful genetic tool to virtually all areas of developmental biol. P-elements are still widely used as traditional mutagenesis reagents and form the backbone of projects aimed at generating insertions in every predicted gene in the fly genome. In addn., vectors based on the FLP/FRT system are being used for genome-wide applications, including the development of molecularly-mapped deletion and duplication kits. In addn. to these 'traditional' genetic approaches, a variety of engineered elements have been developed for a wide range of transgenic applications, including enhancer trapping, gene-tagging, targeted misexpression, RNA interference (RNAi) delivery and ***homologous*** ***recombination*** /gene replacement. To complement the use of P-elements, alternative transposon vectors have been developed. The most widely used of these are the lepidopteran element piggyBac and a Drosophila hydei transposon, Minos. In total, a range of transposon vectors offers the Drosophila biologist considerable flexibility and sophistication in manipulating the genome of the fly and has allowed rapid advances in all areas of developmental biol. and genome science.
RE.CNT 89 THERE ARE 89 CITED REFERENCES AVAILABLE FOR THIS RECORD
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L3 ANSWER 30 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN DUPLICATE 14
AN 2003495582 EMBASE
TI Complexities of chromium carcinogenesis: Role of cellular response, repair and recovery mechanisms.
AU O'Brien T.J.; Ceryak S.; Patierno S.R.
CS S.R. Patierno, Dept. of Pharmacology and Molecular, Cellular Oncology Program, George Washington Univ. Med. Center, 2300 I Street NW, Washington, DC 20037, United States. phmsrp@gwumc.edu
SO Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis,
(10 Dec 2003) 533/1-2 (3-36).
Refs: 330
ISSN: 0027-5107 CODEN: MRFMEC
CY Netherlands
DT Journal; General Review
FS 015 Chest Diseases, Thoracic Surgery and Tuberculosis
016 Cancer
030 Pharmacology
037 Drug Literature Index
052 Toxicology
LA English
SL English
AB Certain hexavalent chromium (Cr(VI))-containing compounds are recognized occupational human lung carcinogens and may pose an environmental health risk. The carcinogenicity of Cr(VI) is targeted to particulate forms of moderate to low solubility. Soluble Cr(VI) oxyanions in the immediate cellular microenvironment traverse the cell membrane by non-specific anionic transporters. Cr(VI) is reductively metabolized within cells by agents including ascorbic acid (Asc), glutathione (GSH) and cysteine (Cys). During Cr(VI) reduction, a diverse range of genetic lesions are generated including Cr-DNA binary (mono) adducts, Cr-DNA ternary adducts, DNA protein crosslinks (DPCs), bi-functional (DNA interstrand crosslinks (ICLs)) adducts, single-strand breaks (SSBs) and oxidized bases. Some forms of Cr damage, such as ICLs, present physical barriers to DNA replication/transcription and, thus, likely promote a terminal cell fate such as apoptosis or terminal growth arrest. Other lesions, such as ternary DNA adducts, are potentially pre-mutagenic. Cr(VI) exposure elicits a classical DNA damage response within cells including activation of the p53 signaling pathway and cell cycle arrest or apoptosis. Moreover, Cr(VI) also induces the ATM-dependent DNA damage response pathway which is paradoxically required for both apoptosis and survival after Cr(VI) insult. In ***yeast*** , moderately cytotoxic concentrations of Cr(VI) result in an initial G1 arrest and delayed S phase progression, whereas less toxic levels of Cr(VI) induce G2 arrest, which requires ***homologous*** ***recombination*** for exit and survival. The past several years has witnessed many important advances in our understanding of the genetic/cellular damage produced by exposure to Cr(VI). Further information is needed regarding the potential involvement of oxygen radicals in Cr genotoxicity, the specific DNA repair pathways activated by Cr and the complex signaling mechanisms involved in the cellular response to Cr(VI). These pertinent issues must be considered in relation to the potential role that each plays in the induction of human respiratory tract cancer by particulate Cr(VI) compounds. .COPYRGT. 2003 Elsevier B.V. All rights reserved.

L3 ANSWER 31 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:139650 CAPLUS
DN 139:229357
TI Studies on the prevention of aerobic spoilage of silage by killer ***yeast*** , Kluyveromyces lactis
AU Kitamoto, Hiroko K.
CS Dep. Gen. Resour. I, Natl. Inst. Agrobiol. Resour., Tsukuba, 305-8602, Japan
SO Nogyo Seibutsu Shigen Kenkyusho Kenkyu Hokoku (2003), 16, 1-72
CODEN: NSSHEC; ISSN: 0911-6575
PB Nogyo Seibutsu Shigen Kenkyusho
DT Journal; General Review
LA English
AB A ***review*** . Aerobic spoilage caused by lactic acid-assimilating ***yeast*** contributes to significant nutritional and dry matter loss

in silage. The author proposes a method of preventing spoilage by use of killer ***yeast*** . The inocula or the crude killer protein of ***yeast*** strain Kluyveromyces lactis IFO1267, effectively prevented the aerobic growth of target ***yeast*** strain in the model system of silage fermn. used. However, this killer ***yeast*** has a possibility to be involved in aerobic spoilage because of its lactic acid-assimilating ability. To construct a killer strain having no ability to grow on lactic acid, the author attempted to disrupt phosphoenolpyruvate carboxykinase (PEPCK) gene by site-directed mutagenesis. PEPCK is one of the key enzymes in gluconeogenesis, which is essential for aerobic growth using lactic acid as a sole carbon source. K. lactis PEPCK gene (KlPCK1) was isolated by complementation of Saccharomyces cerevisiae pck1 mutant. The KlPCK1 defective mutant PCK27 was obtained by ***homologous*** ***recombination*** from K. lactis IFO1267. The strain PCK27 inhibited the growth of target strain and prevented a rise in pH in a model of silage fermn. This suppressive effect of PCK27 was not only due to growth competition but also due to the killer protein produced. From these results, I concluded that strain PCK27 can be used as an additive to prolong the aerobic stability of maize silage. In the lab.-scale expt. of maize silage, the addn. of a killer ***yeast*** changed the ***yeast*** flora and significantly reduced aerobic spoilage.

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L3 ANSWER 32 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
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AN 2003000159 EMBASE

TI Helicase activity is only partially required for Schizosaccharomyces pombe Rqh 1 p function.

AU Ahmad F.; Kaplan C.D.; Stewart E.

CS E. Stewart, School Biological Sciences, University of Manchester, Oxford Road, Manchester MI3 9PT, United Kingdom. elspeth.stewart@man.ac.uk

SO Yeast, (2002) 19/16 (1381-1398).
Refs: 86
ISSN: 0749-503X CODEN: YESTE3

CY United Kingdom

DT Journal; General Review

FS 004 Microbiology

LA English

SL English

AB The RecQ-related family of DNA helicases is required for the maintenance of genomic stability in organisms ranging from bacteria to humans. In humans, mutation of three RecQ-related helicases, BLM, WRN and RecQL4, cause the cancer-prone and premature ageing diseases of Bloom syndrome, Werner's syndrome and Rothmund-Thompson syndrome, respectively. In the fission ***yeast*** Schizosaccharomyces pombe, disruption of the rqh1(+) gene, which encodes the single Sz. pombe RecQ-related helicase, causes cells to display reduced viability and elevated levels of chromosome loss. After S-phase arrest or DNA damage, cells lacking rqh1(+) function display elevated levels of ***homologous*** ***recombination*** and defective chromosome segregation. Here we show that, like other RecQ family members, the Rqh1p protein displays 3' to 5' DNA helicase activity. Interestingly, however, unlike other RecQ family members, the helicase activity of Rqh1p is only partially required for its function in recovery from S-phase arrest or DNA damage. We also report that high cellular levels of Rqh1p result in lethal chromosome segregation defects, while more moderate levels of Rqh1p cause significantly elevated rates of chromosome loss. This suggests that careful regulation of RecQ-like protein levels in eukaryotic cells is vital for maintaining genome stability. Copyright .COPYRGT. 2002 John Wiley & Sons, Ltd.

L3 ANSWER 33 OF 151 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN DUPLICATE 15

AN 2002:546969 BIOSIS

DN PREV200200546969

TI DNA double-strand break repair by ***homologous*** ***recombination*** .

AU van den Bosch, Michael; Lohman, Paul H. M.; Pastink, Albert [Reprint author]

CS Department of Radiation Genetics and Chemical Mutagenesis, Leiden University Medical Center, Wassenaarseweg 72, NL-2333, AL Leiden, Netherlands

SO Biological Chemistry, (June, 2002) Vol. 383, No. 6, pp. 873-892. print.
ISSN: 1431-6730.

DT Article
General Review; (Literature Review)

LA English

ED Entered STN: 23 Oct 2002
Last Updated on STN: 23 Oct 2002

AB The induction of double-strand breaks (DSBs) in DNA by exposure to DNA damaging agents, or as intermediates in normal cellular processes, constitutes a severe threat for the integrity of the genome. If not properly repaired, DSBs may result in chromosomal aberrations, which, in turn, can lead to cell death or to uncontrolled cell growth. To maintain the integrity of the genome, multiple pathways for the repair of DSBs have evolved during evolution: ***homologous*** ***recombination*** (HR), non-homologous end joining (NHEJ) and single-strand annealing (SSA). HR has the potential to lead to accurate repair of DSBs, whereas NHEJ and SSA are essentially mutagenic. In ***yeast*** , DSBs are primarily repaired via high-fidelity repair of DSBs mediated by HR, whereas in higher eukaryotes, both HR and NHEJ are important. In this ***review*** , we focus on the functional conservation of HR from fungi to mammals and on the role of the individual proteins in this process.

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AN 2002247378 EMBASE

TI Recombination functions of replication protein A.

AU Brush G.S.

CS G.S. Brush, Prog. in Molec. Bio. and Genetics, Karmanos Cancer Institute, Wayne State University, 110 E. Warren Ave., Detroit, MI 48201, United States. brushg@karmanos.org

SO Current Organic Chemistry, (2002) 6/9 (795-813).
Refs: 213
ISSN: 1385-2728 CODEN: CORCFE

CY Netherlands

DT Journal; General Review

FS 004 Microbiology
029 Clinical Biochemistry
022 Human Genetics

LA English

SL English

AB Proteins that bind and stabilize single-stranded DNA are critical for proper DNA metabolism. In eukaryotes, the major single-stranded DNA-binding protein is replication protein A (RPA), and evolutionarily conserved heterotrimeric complex required for DNA replication, repair, and recombination. While much of the early work on RPA established its role in DNA replication, a great deal of attention is now being paid to the specific mechanisms by which RPA operates in recombination. As described in this ***review*** , significant insight has been gained from studies employing proteins purified from both ***yeast*** and human cells. Of particular importance, these analyses have revealed that RPA is centrally involved in the initiation of ***homologous*** ***recombination*** . Research into recombination and its influence by RPA is especially to our understanding of disease development, as inappropriate chromosomal rearrangement is known to be associated with a number of human disorders.

L3 ANSWER 35 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2002:351320 CAPLUS

DN 137:165872

TI Sex and the single (double-strand) break

AU Martini, Emmanuelle; Keeney, Scott

CS Molecular Biology Program, Memorial Sloan-Kettering Cancer Center, New York, NY, 10021, USA

SO Molecular Cell (2002), 9(4), 700-702
CODEN: MOCEFL; ISSN: 1097-2765

PB Cell Press

DT Journal; General Review

LA English

AB A ***review*** . It has been known for some time that DNA double-strand breaks (DSBs) initiate ***homologous*** ***recombination*** during meiosis. Two recent studies show that the fate of a single DSB in ***yeast*** is strongly influenced by the presence of other breaks in the genome, hinting that cell-wide or chromosome-regional mechanisms control the outcome of DSB repair.

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L3 ANSWER 36 OF 151 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN DUPLICATE 17

AN 2003:164114 BIOSIS

DN PREV200300164114

TI Formation, repair and detection of DNA double-strand breaks.

AU Brozmanova, Jela [Reprint Author]; Markova, Eva; Dudas, Andrej

CS Cancer Research Institute, Slovak Academy of Sciences, Vlarska 7, SK-83391, Bratislava, Slovakia
exonbroz@savba.sk

SO Biologia (Bratislava), (December 2002) Vol. 57, No. 6, pp. 665-675. print.
CODEN: BLOAAO. ISSN: 0006-3088.

DT Article
General Review; (Literature Review)

LA English

ED Entered STN: 26 Mar 2003
Last Updated on STN: 26 Mar 2003

AB DNA double-strand breaks (DSBs) can be generated exogenously by a variety of genotoxic agents, including ionizing radiation and radiomimetic chemicals. They can also arise endogenously as intermediates during several cellular processes. The repair of DSBs is a complex process that requires multiple enzymatic and structural activities to repair or rejoin the broken DNA ends. There are several systems for elimination of DSBs from DNA in eukaryotes, ***homologous*** ***recombination*** , single-strand annealing and non-homologous end-joining. The ability to repair DSBs is essential for cells, because DSBs inhibit all DNA transactions. Additionally, accumulation of DSBs leads to genomic instability that can result in cancer in higher organisms. Here, we ***review*** current knowledge about molecular mechanisms of the DSB repair. We also describe the main methods for determining the frequency of DSBs in model organisms such as ***yeast*** and mammalian cells.

L3 ANSWER 37 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:800 CAPLUS
DN 138:83907
TI Application of ***yeast*** genetics to biotechnology for producing anti-salinity plant
AU Yoshida, Kazuya
CS Graduate School of Bioscience, Nara Institute of Sciences and Technology, Ikoma-shi, Nara, 630-0101, Japan
SO Seibutsu Kogaku Kaishi (2002), 80(10), 482-485
CODEN: SEKAEA; ISSN: 0919-3758
PB Nippon Seibutsu Kogakkai
DT Journal; General Review
LA Japanese
AB A ***review*** . The technol. of genetic induction of useful ***yeast*** functional genes in plants by using recombination system with MAT (multiauto-transformation) vector was outlined. Prodn. of anti-salinity plant by introducing ***yeast*** genes such as the ENA1 gene for Na-ATPase involved in osmolality regulation was discussed. Some useful osmolality-regulating genes such as HKT1 and HKT2 for K+-Na+ cotransporter were isolated from rice. However, demonstration of physiol. activity of these genes is very difficult since prodn. of gene knockout plant models is difficult for the low ***homologous*** ***recombination*** activity in plants. The use of ***yeast*** gene knockout system for the screening of plant gene function was described to overcome this problem.

L3 ANSWER 38 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:541288 CAPLUS
DN 137:166160
TI A new moss genetics: Targeted mutagenesis in Physcomitrella patens
AU Schaefer, Didier G.
CS Institut d'Ecologie, Laboratoire de Phytogenetique Cellulaire, Universite de Lausanne, Lausanne, CH-1015, Switz.
SO Annual Review of Plant Biology (2002), 53, 477-501
CODEN: ARPBDW
PB Annual Reviews Inc.
DT Journal; General Review
LA English
AB A ***review*** . The potential of moss as a model system to study plant biol. is assocd. with a relatively simple developmental pattern that nevertheless resembles the basic organization of the body plan of land plants, the direct access to cell-lineage anal., their similar responses to plant growth factors and environmental stimuli as those obsd. in other land plants, and the dominance of the gametophyte in the life cycle that facilitates genetic approaches. Transformation studies in the moss Physcomitrella patens have revealed a totally unique feature for plants, i.e., that foreign DNA sequences integrate in the genome preferentially at targeted locations by ***homologous*** ***recombination*** , enabling for the first time in plants the application of the powerful mol. genetic approaches used routinely in bacteria, ***yeast*** , and since 1989, the mouse embryonic stem cells. This article reviews our current knowledge of Physcomitrella patens transformation and its unique suitability for functional genomic studies.
RE.CNT 134 THERE ARE 134 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 39 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:801325 CAPLUS
DN 138:199250
TI ***Homologous*** ***recombination*** : ends as the means
AU Ray, Animesh; Langer, Marybeth
CS Keck Graduate Institute, Claremont, CA, 91711, USA
SO Trends in Plant Science (2002), 7(10), 435-440
CODEN: TPSCF9; ISSN: 1360-1385
PB Elsevier Science Ltd.
DT Journal; General Review
LA English
AB A ***review*** . Broken chromosomal ends in somatic cells of higher plants frequently heal by the ligation of DNA ends to unrelated sequences or to sequences with micro-homologies. This pathway of DNA-strand-break repair is the bane of gene-targeting attempts in plants. However, there is a second somatic pathway of chromosome repair, which is driven by DNA-sequence homol. Observations from ***yeast*** , fly and plants of ***homologous*** - ***recombination*** mechanisms point towards new strategies of gene targeting in plants. Observations from ***yeast*** , fly and plants on ***homologous*** ***recombination*** mechanisms point towards new strategies of gene targeting in plants.
RE.CNT 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 40 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:738299 CAPLUS
DN 140:105843
TI Advances in eukaryotic expression systems
AU Gao, Yun
CS Laboratory of Reproduction and Genetics, Nanjing General Hospital of Nanjing Command, PLA, Nanjing, Jiangsu Province, 210002, Peop. Rep. China
SO Zhonghua Nankexue (2002), 8(4), 292-294, 298
CODEN: ZNHAAT; ISSN: 1009-3591
PB Zhonghua Nankexue Bianjibu
DT Journal; General Review
LA Chinese
AB A ***review*** . The increasing popularity of eukaryotic expression systems can be attributed to their capability of performing many post-translational modifications. At present, there are mainly three expression systems including ***yeast*** expression system, insect cell expression system and mammalian cell expression system. The methylotropic ***yeast*** Pichia Pastoris usually utilizes alc. oxidase promoter to drive the expression of foreign gene. Recently, a continuous fermn. has been developed in Pichia Pastoris with the glyceraldehyde-3-phosphate dehydrogenase (GAP) promoter. The baculovirus-mediated insect cell expression system is considered to be safe, powerful, but cell-lytic. Baculovirus -S2 system uses the popular and genetically well understood Drosphila S2 cells which do not appear to be lysed after infection. In mammalian cell expression system, recombinant adenovirus are attracting a great deal of attention as a highly efficient gene transfer vehicle. The frequency of Ad vector rescue by ***homologous*** ***recombination*** in E. coli and Cre-mediated site-specific recombination is significantly higher than by ***homologous*** ***recombination*** in vivo. Tetracycline-regulatable system is a widely used mammalian cell inducible expression system due to its high efficiency and stringency.

L3 ANSWER 41 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:126944 CAPLUS
DN 137:42158
TI Gene replacement by ***homologous*** ***recombination*** in plants
AU Puchta, Holger
CS Institut fur Pflanzengenetik und Kulturpflanzenforschung (IPK), Gatersleben, 06466, Germany
SO Plant Molecular Biology (2002), 48(1-2), 173-182
CODEN: PMBIDB; ISSN: 0167-4412
PB Kluwer Academic Publishers
DT Journal; General Review
LA English
AB A ***review*** . After the elucidation of the sequence of the ***yeast*** genome, a major effort was started to elucidate the biol. function of all open reading frames of this organism by targeted gene replacement via ***homologous*** ***recombination*** . The establishment of the complete sequence of the genome of Arabidopsis thaliana would principally allow a similar approach. However, over the past dozen years all attempts to establish an efficient gene targeting technique in flowering plants have been unsuccessful. In contrast, in Physcomitrella patens an efficient gene targeting procedure has been set up, making the moss a valuable model system for plant mol. biologists. In flowering plants several new approaches - some of them based on the availability of the genomic sequence of Arabidopsis - were initiated recently that might finally result in a general applicable technique. The application of chimeric oligonucleotides, which can produce hyper-recombinogenic plants either via expression or suppression of specific gene functions or via undirected mutagenesis, may result in major progress.
RE.CNT 81 THERE ARE 81 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 42 OF 151 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 18
AN 2003:138538 BIOSIS
DN PREV200300138538
TI Control of meiotic recombination initiation: A role for the environment?.
AU Koren, Amnon; Ben-Aroya, Shay; Kupiec, Martin [Reprint Author]
CS Department of Molecular Microbiology and Biotechnology, Tel Aviv University, Ramat Aviv, 69978, Israel
martin@post.tau.ac.il
SO Current Genetics, (December 2002) Vol. 42, No. 3, pp. 129-139. print. ISSN: 0172-8083 (ISSN print).
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 12 Mar 2003
Last Updated on STN: 12 Mar 2003
AB ***Homologous*** ***recombination*** plays a central role during meiosis, ensuring the proper segregation of homologous chromosomes during the first meiotic division. In addition, meiotic recombination generates genetic variability upon which natural selection can act. The frequency of recombination is not evenly distributed throughout the genome: regions of high (hotspots) and low (coldspots) recombination can be found. Meiotic hotspots exhibit high levels of double-strand break formation and these breaks coincide with the upstream regions of genes. In many cases, binding of transcription factors has been shown to be required for hotspot activity. We ***review*** the current knowledge on the mechanisms that determine hotspot activity and propose a modified model to account for recent observations which show that recombination frequency at hotspots is sensitive to environmental conditions.

L3 ANSWER 43 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:563370 CAPLUS
DN 137:307060
TI Genetic diversity of yeasts in wine production
AU Benitez, Tahia; Codon, Antonio C.
CS Department of Genetics, Faculty of Biology, University of Seville, Seville, E-41080, Spain

SO Applied Mycology and Biotechnology (2002), 2(Agriculture and Food Production), 19-44
CODEN: AMBPC2
PB Elsevier Science B.V.
DT Journal; General Review
LA English
AB A ***review*** . Wine elaboration is a complex multipopulational process in which several microbial species are successively involved. At the early stages of fermn., a high no. of non-Saccharomyces ***yeast*** species predominate in the musts. Although Saccharomyces cerevisiae wine yeasts showed polymorphisms of their chromosomes, under extreme conditions strains showed an almost unique chromosomal pattern while restriction fragment length polymorphism of their mtDNA was highly variable. This polymorphism appears to result from chromosome reorganizations, ***homologous*** ***recombination*** and gene conversion, occurring both at mitosis and meiosis and, in some cases, mediated by the presence of DNA repeats such as Y' or X subtelomeric regions or Ty transposable elements. Reorganizations and changes in DNA sequences might be favored by DNA breaks caused by ethano and DNA repair via recombination.
RE.CNT 105 THERE ARE 105 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 44 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN
AN 2002352737 EMBASE
TI Gene targeting by ***homologous*** ***recombination*** : A powerful addition to the genetic arsenal for Drosophila geneticists.
AU Rong Y.S.
CS Y.S. Rong, Laboratory of Molecular Cell Biology, National Cancer Institute, NIH, 37 Convent Dr., Bethesda, MD 20892, United States.
rongy@mail.nih.gov
SO Biochemical and Biophysical Research Communications, (2002) 297/1 (1-5).
Refs: 33
ISSN: 0006-291X CODEN: BBRCA
PUI S 0006-291X(02)02066-1
CY United States
DT Journal; General Review
FS 029 Clinical Biochemistry
LA English
SL English
AB A series of recent publications have firmly established the notion that Drosophila researchers now have a general method to subject genes for targeted modification by ***homologous*** ***recombination*** (HR) [Science 288 (2000) 2013; Genetics 157 (3) (2001) 1307; Genes Dev. 16 (12) (2002) 1568; Genetics 161 (2002) 1125-1136]. This method allows one to knockout essentially any gene starting with the DNA sequence of the gene. It has greatly enhanced studies of gene function as demonstrated by over 20 years of gene targeting practice in ***yeast*** and mouse. Here, I discuss the basic targeting methodology for eukaryotic organisms. I compare the Drosophila method with the traditional targeting scheme in ***yeast*** and mouse mainly to show that the targeting mechanism as well as many aspects of the experimental design remain unchanged, and that the Drosophila scheme differs only in the way in which the donor molecule for targeting is generated. I propose that the Drosophila method can be readily adapted in other organisms without culturable stem cells, since the mechanism for in vivo donor generation in Drosophila is likely to be functional in a variety of different organisms. .COPYRGT. 2002 Elsevier Science (USA). All rights reserved.

L3 ANSWER 45 OF 151 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN DUPLICATE 19
AN 2001:397214 BIOSIS
DN PREV200100397214
TI Meiotic recombination and chromosome segregation in Schizosaccharomyces pombe.
AU Davis, Luther; Smith, Gerald R. [Reprint author]
CS Fred Hutchinson Cancer Research Center, 1100 Fairview Avenue North, A1-162, Seattle, WA, 98109-1024, USA
gsmith@fhcrc.org
SO Proceedings of the National Academy of Sciences of the United States of America, (July 17, 2001) Vol. 98, No. 15, pp. 8395-8402. print.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 22 Aug 2001
Last Updated on STN: 22 Feb 2002
AB In most organisms ***homologous*** ***recombination*** is vital for the proper segregation of chromosomes during meiosis, the formation of haploid sex cells from diploid precursors. This ***review*** compares meiotic recombination and chromosome segregation in the fission ***yeast*** Schizosaccharomyces pombe and the distantly related budding ***yeast*** Saccharomyces cerevisiae, two especially tractable microorganisms. Certain features, such as the occurrence of DNA breaks associated with recombination, appear similar, suggesting that these features may be common in eukaryotes. Other features, such as the role of these breaks and the ability of chromosomes to segregate faithfully in the absence of recombination, appear different, suggesting multiple solutions to the problems faced in meiosis.

L3 ANSWER 46 OF 151 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN DUPLICATE 20
AN 2001:396820 BIOSIS
DN PREV200100396820
TI Homologous DNA recombination in vertebrate cells.
AU Sonoda, Eiichiro; Takata, Minoru; Yamashita, Yukiko M.; Morrison, Ciaran; Takeda, Shunichi [Reprint author]
CS Department of Radiation Genetics, Faculty of Medicine, Kyoto University, Yoshida Konoe, Sakyo-ku, Kyoto, 606-8501, Japan
stakeda@rg.med.kyoto-u.ac.jp
SO Proceedings of the National Academy of Sciences of the United States of America, (July 17, 2001) Vol. 98, No. 15, pp. 8388-8394. print.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 22 Aug 2001
Last Updated on STN: 22 Feb 2002
AB The RAD52 epistasis group genes are involved in homologous DNA recombination, and their primary structures are conserved from ***yeast*** to humans. Although biochemical studies have suggested that the fundamental mechanism of homologous DNA recombination is conserved from ***yeast*** to mammals, recent studies of vertebrate cells deficient in genes of the RAD52 epistasis group reveal that the role of each protein is not necessarily the same as that of the corresponding ***yeast*** gene product. This ***review*** addresses the roles and mechanisms of ***homologous*** ***recombination*** -mediated repair with a special emphasis on differences between ***yeast*** and vertebrate cells.

L3 ANSWER 47 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN DUPLICATE 21
AN 2001235368 EMBASE
TI Spectrin and ankyrin-based pathways: Metazoan inventions for integrating cells into tissues.
AU Bennett V.; Baines A.J.
CS V. Bennett, Howard Hughes Medical Institute, Dept. of Cell Biology, Duke University Medical Center, Durham, NC 27710, United States.
benne012@mc.duke.edu
SO Physiological Reviews, (2001) 81/3 (1353-1392).
Refs: 455
ISSN: 0031-9333 CODEN: PHREA7
CY United States
DT Journal; General Review
FS 029 Clinical Biochemistry
LA English
SL English
AB The spectrin-based membrane skeleton of the humble mammalian erythrocyte has provided biologists with a set of interacting proteins with diverse roles in organization and survival of cells in metazoan organisms. This ***review*** deals with the molecular physiology of spectrin, ankyrin, which links spectrin to the anion exchanger, and two spectrin-associated proteins that promote spectrin interactions with actin: adducin and protein 4.1. The lack of essential functions for these proteins in generic cells grown in culture and the absence of their genes in the ***yeast*** genome have, until recently, limited advances in understanding their roles outside of erythrocytes. However, completion of the genomes of simple metazoans and application of ***homologous*** ***recombination*** in mice now are providing the first glimpses of the full scope of physiological roles for spectrin, ankyrin, and their associated proteins. These functions now include targeting of ion channels and cell adhesion molecules to specialized compartments within the plasma membrane and endoplasmic reticulum of striated muscle and the nervous system, mechanical stabilization at the tissue level based on transcellular protein assemblies, participation in epithelial morphogenesis, and orientation of mitotic spindles in asymmetric cell divisions. These studies, in addition to stretching the erythrocyte paradigm beyond recognition, also are revealing novel cellular pathways essential for metazoan life. Examples are ankyrin-dependent targeting of proteins to excitable membrane domains in the plasma membrane and the Ca(2+) homeostasis compartment of the endoplasmic reticulum. Exciting questions for the future relate to the molecular basis for these pathways and their roles in a clinical context, either as the basis for disease or more positively as therapeutic targets.

L3 ANSWER 48 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:404308 CAPLUS
DN 135:56544
TI Homologous DNA recombination is essential for the proliferation of vertebrate cells
AU Sonoda, Eiichiro; Takata, Minoru; Yamashita, Yukiko M.; Takeda, Shunichi
CS Grad. Sch. Med., Kyoto Univ., Japan
SO Tanpakushitsu Kakusan Koso (2001), 46(8, 6gatsuzokan), 1046-1054
CODEN: TAKKAJ; ISSN: 0039-9450
PB Kyoritsu Shuppan
DT Journal; General Review
LA Japanese
AB A ***review*** with 21 refs., on the comparison of DNA repair systems between ***yeast*** and animal cells, usefulness of chicken DT cells in the anal. of DNA repair genes, necessity of DNA repair by ***homologous*** ***recombination*** in animal cell proliferation,

and differences in homologous DNA recombination mechanisms between ***yeast*** and vertebrate cells. Mechanisms of DNA repair of double-strand breaks, phenotypes of Rad51-, Rad51 paralog-, Rad52-, or Rad54-deficient cells, homol. of Rad51 paralogs and Rad52, functions of BRCA1 and BRCA2 in vertebrate cells, and involvement of Mre11/Rad50/Nbs1 (Xrs2) complexes in DNA damage checkpoint are discussed.

L3 ANSWER 49 OF 151 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:404306 CAPLUS
DN 135:56543
TI Functional regulation of the Mre11/Rad50/Xrs2 complex in the processes of recombination and DSB repair
AU Usui, Takehiko; Ogawa, Tomoko; Ogawa, Hideyuki
CS Madison Coll., Univ. Wisconsin, USA
SO Tanpakushitsu Kakusan Koso (2001), 46(8, 6gatsuzokan), 1030-1037
CODEN: TAKKAJ; ISSN: 0039-9450
PB Kyoritsu Shuppan
DT Journal; General Review
LA Japanese
AB A ***review*** with 54 refs., on the structural characteristics of Mre11, Rad50, and Xrs2 of budding ***yeast*** , roles of Mre11 protein in the initiation of meiotic recombination and DNA double-strand break (DSB) formation, involvement of Mre11/Rad50/Xrs2 complex in ***homologous*** ***recombination*** and non-homologous end-joining, DSB processing by Mre11 complex, assocn. of MRE11 complex and ATM (ataxia
telangiectasia mutated) with DNA damage checkpoint in humans, involvement of mutations in MRE11 complex in Nijmegen breakage syndrome and ataxia telangiectasia like disorder, and role of MRE11 complex in telomere maintenance.

L3 ANSWER 50 OF 151 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
RESERVED. on STN
AN 2001200460 EMBASE
TI ***Homologous*** ***recombination*** : From model organisms to human disease.
AU Modesti M.; Kanaar R.
CS R. Kanaar, Department of Cell Biology/Genetics, Erasmus University Rotterdam, 3000 DR Rotterdam, Netherlands. kanaar@gen.fgg.eur.nl
SO Genome Biology, (2001) 2/5 (1014.1-1014.5).
Refs: 32
ISSN: 1465-6906 CODEN: GNBLFW
CY United Kingdom
DT Journal; General Review
FS 004 Microbiology
016 Cancer
022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
AB Recent experiments show that properly controlled recombination between homologous DNA molecules is essential for the maintenance of genome stability and for the prevention of tumorigenesis.

=> d his

(FILE 'HOME' ENTERED AT 14:36:07 ON 02 SEP 2004)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 14:38:10 ON 02 SEP 2004
L1 2137 S HOMOLOGOUS RECOMBINATION AND YEAST
L2 220 S L1 AND REVIEW
L3 151 DUP REM L2 (69 DUPLICATES REMOVED)

=> s l1 and targeting construct
L4 8 L1 AND TARGETING CONSTRUCT

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 4 DUP REM L4 (4 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L5 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:991689 CAPLUS
DN 140:54476
TI In vivo production of retrons for gene targeting
IN Rozwadowski, Kevin L.; Lydiate, Derek J.
PA Her Majesty In Right of Canada as Represented by the Minister of Agriculture and Agri-Food Canada, Can.
SO PCT Int. Appl., 201 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003104470	A2	20031218	WO 2003-CA850	20030605
WO 2003104470	A3	20040610		

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2002-386640P P 20020605
AB The invention provides methods and nucleic acid constructs that may be used to modify a nucleic acid of interest at a target locus within the genome of a host. In some aspects, the invention contemplates producing in vivo a gene targeting substrate (GTS), which may be comprised of both DNA and RNA components. The gene targeting substrate may comprise a gene
targeting nucleotide sequence (GTNS), which is homologous to the target locus, but comprises a sequence modification compared to the target locus. The invention relates to systems for producing gene targeting substrates using RNA intermediates and methods for promoting in vivo gene modification using such gene targeting substrates. The invention claims a method to modify a nucleic acid at a target locus within the genome of a host by introducing a gene ***targeting*** ***construct*** (GTC) into the host. The GTC may be a DNA sequence integrated into the genome of the host, or integrated into an extrachromosomal element. The GTC may be used to produce a gene targeting mRNA by transcription. The gene targeting mRNA is capable of folding or hybridizing to form a primer for reverse transcriptase. Reverse transcription then produces the gene targeting substrate. The host expressing the GTC is modified to be capable of expressing a reverse transcriptase at the same time, or after, the host expresses the GTC. The structure of retrons may be adapted for use in the gene targeting systems of the invention. Following expression of the gene targeting systems of the invention, hosts may for example be selected having genomic modifications at a target locus that correspond to the sequence modification present on the gene targeting nucleotide sequence. An example of the invention shows expression of cDNAs in Saccharomyces cerevisiae from derivs. of msr-msd retron elements having different size inserts. CDNA expression depends on the presence of a plasmid vector encoding reverse transcriptase with a nuclear localization sequence. Gene targeting cassettes were shown to convert the chromosomal URA3 gene of S. cerevisiae to a non-functional allele (ura3).

L5 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:794235 CAPLUS
DN 137:274176
TI ***Homologous*** ***recombination*** in mismatch repair inactivated eukaryotic cells
IN Te Riele, Henricus Petrus Joseph; De Wind, Niels; Dekker-Vlaar, Helena Maria Johanna
PA Neth.
SO U.S. Pat. Appl. Publ., 18 pp., Cont.-in-part of U.S. Ser. No. 147,712, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002151059	A1	20021017	US 2001-884877	20010620
WO 9705268	A1	19970213	WO 1995-EP2980	19950726
US 2003221208	A1	20031127	US 2003-365312	20030212

W: AU, BR, CA, CN, JP, KR, MX, NO, NZ, RU, SE, SG, US
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRAI WO 1995-EP2980 W 19950726
US 1999-147712 B2 19990223
US 2001-884877 A3 20010620
AB A mammalian cell having a mismatch repair-deficient phenotype is provided, where one or both alleles of a gene essential for mismatch repair, such as an Msh gene, are inactivated. Using this cell in a gene knock-out methodol. advantageously allows efficient ***homologous*** ***recombination*** , even when the DNA sequences of the donor and recipient sequences diverge by significantly more than 0.6%. The present invention relates to a method for modifying the genome of eukaryotic cells by ***homologous*** ***recombination*** using DNA sequences which substantially differ from the target locus with respect to the nucleotide sequence (0.1 to 30 % divergence) in the region where recombination takes place. ***Homologous*** ***recombination*** between diverged sequences is achieved by genetic or transitory inactivation of the cell's mismatch repair system.

L5 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 1
AN 1998:271074 BIOSIS
DN PREV199800271074
TI Modification of bacterial artificial chromosomes through Chi-stimulated ***homologous*** ***recombination*** and its application in zebrafish transgenesis.
AU Jessen, Jason R.; Meng, Anming; McFarlane, Ramsay J.; Paw, Barry H.; Zon, Leonard I.; Smith, Gerald R.; Lin, Sho [Reprint author]
CS Inst. Molecular Med. Genetics, Med. Coll. Georgia, 1120 15th St., Augusta, GA 30912, USA

SO Proceedings of the National Academy of Sciences of the United States of America, (April 28, 1998) Vol. 95, No. 9, pp. 5121-5126. print.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 24 Jun 1998
Last Updated on STN: 24 Jun 1998
AB The modification of ***yeast*** artificial chromosomes through ***homologous*** ***recombination*** has become a useful genetic tool for studying gene function and enhancer/promoter activity. However, it is difficult to purify intact ***yeast*** artificial chromosome DNA at a concentration sufficient for many applications. Bacterial artificial chromosomes (BACs) are vectors that can accommodate large DNA fragments and can easily be purified as plasmid DNA. We report herein a simple procedure for modifying BACs through ***homologous*** ***recombination*** using a ***targeting*** ***construct*** containing properly situated Chi sites. To demonstrate a usage for this technique, we modified BAC clones containing the zebrafish GATA-2 genomic locus by replacing the first coding exon with the green fluorescent protein (GFP) reporter gene. Molecular analyses confirmed that the modification occurred without additional deletions or rearrangements of the BACs. Microinjection demonstrated that GATA-2 expression patterns can be recapitulated in living zebrafish embryos by using these GFP-modified GATA-2 BACs. Embryos microinjected with the modified BAC clones were less mosaic and had improved GFP expression in hematopoietic progenitor cells compared with smaller plasmid constructs. The precise modification of BACs through Chi-stimulated ***homologous*** ***recombination*** should be useful for studying gene function and regulation in cultured cells or organisms where gene transfer is applicable.

L5 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 2
AN 1995:124575 BIOSIS
DN PREV199598138875
TI Repair of a specific double-strand break generated within a mammalian chromosome by ***yeast*** endonuclease I-SceI.
AU Lukacsovich, Tamas; Yang, Di; Waldman, Alan S. [Reprint author]
CS Dep. Biol. Sci., Univ. S.C., Columbia, SC 29208, USA
SO Nucleic Acids Research, (1994) Vol. 22, No. 25, pp. 5649-5657.
CODEN: NARHAD. ISSN: 0305-1048.
DT Article
LA English
ED Entered STN: 29 Mar 1995
Last Updated on STN: 23 May 1995
AB We established a mouse Ltk- cell line that contains within its genome a herpes simplex virus thymidine kinase gene (tk) that had been disrupted by the insertion of the recognition sequence for ***yeast*** endonuclease I-SceI. The artificially introduced 18 bp I-SceI recognition sequence was likely a unique sequence in the genome of the mouse cell line. To assess whether an induced double-strand break (DSB) in the genomic tk gene would be repaired preferentially by gene targeting or non- ***homologous*** ***recombination*** , we electroporated the mouse cell line with endonuclease I-SceI alone, one of two different gene targeting constructs alone, or with I-SceI in conjunction with each of the two targeting constructs. Each ***targeting*** ***construct*** was, in principle, capable of correcting the defective genomic tk sequence via ***homologous*** ***recombination*** . tk+ colonies were recovered following electroporation of cells with I-SceI in the presence or absence of a ***targeting*** ***construct*** . Through the detection of small deletions at the I-SceI recognition sequence in the mouse genome, we present evidence that a specific DSB can be introduced into the genome of a living mammalian cell by ***yeast*** endonuclease I-SceI. We further report that a DSB in the genome of a mouse Ltk- cell is repaired preferentially by non-homologous end-joining rather than by targeted ***homologous*** ***recombination*** with an exogenous donor sequence. The potential utility of this system is discussed.

=> s l1 and YTC
2 FILES SEARCHED...
L6 2 L1 AND YTC

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y/(N):y

L6 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:376305 CAPLUS
DN 138:380408
TI Production of gene targeting vectors using ***homologous*** ***recombination*** in ***yeast*** and use of gene targeting vectors in mice
IN Fisher, Katherine E.; Reaume, Andrew G.
PA USA
SO U.S. Pat. Appl. Publ., 20 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003092183	A1	20030515	US 2001-961163	20010921
PRAI US 2001-961163		20010921		

AB The present invention provides methods of prepg. gene targeted mammalian cells having a targeted gene mutation, methods of making gene targeted mice, and gene targeting vectors that are useful in these methods. The method of prepg. gene targeting vectors uses ***homologous*** ***recombination*** in ***yeast*** to screen for genomic clones of interest and to replace a defined portion of a gene of interest with a pos. selection marker that can be used in both Escherichia coli and mammalian cell culture. Specifically, the invention comprises transforming ***yeast*** cells with a RKO clone and a ***yeast*** targeting cassette (***YTC***). The RKO clone has a genomic insert, a ***yeast*** replication element, a ***yeast*** selectable marker, a bacterial origin of replication, a bacterial selectable marker, and a mammalian neg. selection marker. The ***YTC*** contains a bacterial/mammalian pos. selection marker flanked by recombinogenic arms. The RKO clone and ***YTC*** undergo ***homologous*** ***recombination*** to produce a gene targeting vector which can be selected in ***yeast*** and transformed bacteria. When mammalian cells are transformed with the gene targeting vector, ***homologous*** ***recombination*** between genomic DNA and the gene targeting vector produces a targeted gene mutation. The gene targeted mammalian cells are selected by expression of a bacterial/mammalian pos. selection marker. A mammalian neg. selection marker is sepd. from the pos. selection marker in the gene targeting vector during the recombination process. Gene targeted mice are produced by transformation of embryonic stem cells with the gene targeting vector.

L6 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:169183 CAPLUS
DN 136:211850
TI Construction of gene knockout vectors using RKO clone and ***yeast*** targeting cassette ***homologous*** ***recombination*** in ***yeast***
IN Fisher, Katherine Elizabeth; Reaume, Andrew Gerard
PA Pfizer Products Inc., USA
SO Eur. Pat. Appl., 20 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1184461	A2	20020306	EP 2001-307021	20010817
EP 1184461	A3	20020522		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002291471	A2	20021008	JP 2001-250528	20010821
PRAI US 2000-228467P	P	20000825		

AB The present invention discloses methods of developing gene-targeting vectors including transforming ***yeast*** cells with a RKO clone and a ***yeast*** targeting cassette, ***homologous*** ***recombination*** in ***yeast*** , and a subsequent seletion in E. coli, as well as uses of the vectors for directed mutation in a target gene, preferred in embryonic stem cells or whole animals. In particular, the invention discloses that the RKO clone comprises a genomic clone insert, a ***yeast*** replication element, a ***yeast*** selectable marker, a bacterial origin of replication, and a bacterial mammalian pos. selection marker, where the ***YTC*** comprises a bacterial/mammalian pos. seletion marker flanked by recombinogenic arms. The invention also provides compns. and methods for prepg. gene targeted mammalian cells and gene knockout mice.

=> s l1 and target?
L7 470 L1 AND TARGET?

=> d bib abs

L7 ANSWER 1 OF 470 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN
AN 2004:214233 BIOSIS
DN PREV200400212283
TI Maximizing the potential of functional genomics.
AU Steinmetz, Lars M. [Reprint Author]; Davis, Ronald W.
CS European Molecular Biology Laboratory, Meyerhofstrasse 1, 69117, Heidelberg, Germany
lars.steinmetz@embl.de
SO Nature Reviews Genetics, (March 2004) Vol. 5, No. 3, pp. 190-201. print.
ISSN: 1471-0056 (ISSN print).
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

=> s l7 and knockout
L8 30 L7 AND KNOCKOUT

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9 21 DUP REM L8 (9 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 21 ANSWERS - CONTINUE? Y/(N):y

L9 ANSWER 1 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2004:319315 CAPLUS
DN 141:48238
TI Construction of a complete URA5 deletion strain of a human pathogenic ***yeast*** Cryptococcus neoformans
AU Drivinya, Antra; Shimizu, Kiminori; Takeo, Kanji
CS Research Center for Pathogenic Fungi and Microbial Toxicoses, Chiba University, Chuo-ku, Chiba, 260-8673, Japan
SO Nippon Ishinkin Gakkai Zasshi (2004), 45(1), 1-6
CODEN: NIGZE4; ISSN: 0916-4804
PB Nippon Ishinkin Gakkai
DT Journal
LA English
AB Cryptococcus neoformans is an opportunistic human pathogen, which infects the central nervous system causing the fatal disease, meningitis. In order to understand the genetic background of this human pathogen, the basic mol. manipulation techniques of deletion, overexpression, and so on have been developed. URA5, a gene encoding orotate phosphoribosyltransferase, has frequently been used to introduce foreign gene fragments by complementing ura5 mutant strains, which are not, however, stable; reversion to uracil prototroph is thus frequently obsd. on selective condition. The high possibility of reversion makes it inconvenient to use this mutation to identify appropriate transformants and thus, manipulation in mol. genetics. We report here the isolation of a stable ura5 mutant of C. neoformans, designated as TAD1, by eliminating the URA5 gene by ***homologous*** ***recombination*** using the biolistic DNA delivery system. The availability of the stable ura5 mutant offers the advantage that no spontaneous reversion occurs so that a satisfactory rate of ***homologous*** ***recombination*** can be achieved. The strain will allow efficient genomic anal. in C. neoformans.
RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 21 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 1
AN 2004244882 EMBASE
TI ***Targeted*** mutagenesis of the Sap47 gene of Drosophila: Flies lacking the synapse associated protein of 47 kDa are viable and fertile.
AU Funk N.; Becker S.; Huber S.; Brunner M.; Buchner E.
CS E. Buchner, Lehrstuhl fur Genetik/Neurobiologie, Am Hubland, D-97074 Wurzburg, Germany. buchner@biozentrum.uni-wuerzburg.de
SO BMC Neuroscience, (29 Apr 2004) 5/-.
Refs: 24
ISSN: 1471-2202 CODEN: BNMEA6
CY United Kingdom
DT Journal; Article
FS 008 Neurology and Neurosurgery
029 Clinical Biochemistry
LA English
SL English
AB Background: Conserved proteins preferentially expressed in synaptic terminals of the nervous system are likely to play a significant role in brain function. We have previously identified and molecularly characterized the Sap47 gene which codes for a novel synapse associated protein of 47 kDa in Drosophila. Sequence comparison identifies homologous proteins in numerous species including C. elegans, fish, mouse and human. First hints as to the function of this novel protein family can be obtained by generating mutants for the Sap47 gene in Drosophila. Results: Attempts to eliminate the Sap47 gene through ***targeted*** mutagenesis by ***homologous*** ***recombination*** were unsuccessful. However, several mutants were generated by transposon remobilization after an appropriate insertion line had become available from the Drosophila P-element screen of the Bellen/Hoskins/Rubin/Spradling labs. Characterization of various deletions in the Sap47 gene due to imprecise excision of the P-element identified three null mutants and three hypomorphic mutants. Null mutants are viable and fertile and show no gross structural or obvious behavioural deficits. For cell-specific over-expression and "rescue" of the knock-out flies a transgenic line was generated which expresses the most abundant transcript under the control of the ***yeast*** enhancer UAS. In addition, knock-down of the Sap47 gene was achieved by generating 31 transgenic lines expressing Sap47 RNAi constructs, again under UAS control. When driven by a ubiquitously expressed ***yeast*** transcription factor (GAL4), Sap47 gene suppression in several of these lines is highly efficient resulting in residual SAP47 protein concentrations in heads as low as 6% of wild type levels. Conclusion: The conserved synaptic protein SAP47 of Drosophila is not essential for basic synaptic function. The Sap47 gene region may be refractory to ***targeted*** mutagenesis by ***homologous*** ***recombination*** . RNAi using a construct linking genomic DNA to anti-sense cDNA in our hands is not more effective than using a cDNA-anti-sense cDNA construct. The tools developed in this study will now allow a detailed analysis of the molecular, cellular and systemic function of the SAP47 protein in Drosophila. .COPYRGT. 2004 Funk et al, licensee BioMed Central Ltd.

L9 ANSWER 3 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:301230 CAPLUS
DN 138:298827
TI Transposon mediated double positive selection vector for gene ***targeting*** or ***homologous*** ***recombination***
IN Morrison, John; Zhang, Chunfang
PA Copyrat Pty. Ltd., Australia
SO PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003031629	A1	20030417	WO 2002-AU1367	20021008
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1451330	A1	20040901	EP 2002-800517	20021008
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRAI AU 2001-8174	A	20011009		
AU 2002-2522	A	20020523		
WO 2002-AU1367	W	20021008		

AB The present invention relates to providing methods for prepg. a ***targeting*** vector for gene ***targeting*** or ***homologous*** ***recombination*** . The invention also provides ***targeting*** vectors, and cells, plants and animals (including ***yeast***) contg. the vectors having predetd. modifications. The invention further provides plants and animals modified by the ***targeting*** vectors. The gene ***targeting*** methods used herein are based on transposon and recombination mediated procedures, such as Cre-loxP recombinase system, which provide for high throughput generation of deletions. The method was used to ***knockout*** rat HPRT gene.
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:17760 CAPLUS
DN 138:84425
TI Bacteria- ***yeast*** shuttle vectors and methods for preparing mouse genomic libraries for ***knockout*** ***targeting*** vectors
IN Thukral, Sushil K.
PA Amgen Inc., USA
SO U.S., 25 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6503712	B1	20030107	US 2000-569975	20000510
US 2003104456	A1	20030605	US 2002-291022	20021108
PRAI US 2000-569975	A3	20000510		

AB The present invention is directed to methods for producing gene ***targeting*** constructs by ***homologous*** ***recombination*** using mouse genomic libraries arrayed in ***yeast*** shuttle vectors. The present invention provides methods of prepg. a genomic library for use in producing ***knockout*** ***targeting*** vectors comprising prepg. a size selected mouse genomic DNA; prepg. a shuttle vector by inserting said genomic DNA into vector pYYL-1, wherein the vector comprises a bacterial origin of replication, a bacterial selection marker, a ***yeast*** origin of replication, a ***yeast*** selection marker, and a mammalian selection marker; introducing the resulting vectors into bacterial host cells; and arraying the transformed bacteria into pools. The genomic DNA is a library which comprises mouse genomic DNA fragments ranging from about 8 kb to about 14 kb. In this manner, ***knockout*** vectors for mouse genes GPR-24 and CHL-1 were prepd.
RE.CNT 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:660984 CAPLUS
DN 139:302695
TI New "marker swap" plasmids for converting selectable markers on budding ***yeast*** gene disruptions and plasmids
AU Voth, Warren P.; Jiang, Yi Wei; Stillman, David J.
CS Department of Pathology University of Utah, Salt Lake City, UT, 84132, USA
SO Yeast (2003), 20(11), 985-993
CODEN: YESTE3; ISSN: 0749-503X
PB John Wiley & Sons Ltd.
DT Journal
LA English
AB Marker swap plasmids can be used to change markers for genes disrupted with nutritional markers in the ***yeast*** Saccharomyces cerevisiae.

We describe 18 new marker swap plasmids, and we also review other plasmids available for marker conversions. All of these plasmids have long regions of flanking sequence identity, and thus the efficiency of ***homologous*** ***recombination*** mediated by marker conversion is very high. Marker swaps allow one to easily perform crosses to construct double mutant strains even if each of the disrupted strains contains the same marker, as is the case with the KanMX marker used in the ***yeast*** ***knockout*** collection. Marker swaps can also be used to change the selectable marker on plasmids, eliminating the need for subcloning.
RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 6 OF 21 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2003082032 EMBASE
TI Current successes and limitations of using genetic modification for blood pressure research.
AU Mullins L.J.; Mullins J.
CS L.J. Mullins, Molecular Physiology Laboratory, Univ. of Edinburgh Medical School, Teviot Place, Edinburgh, EH8 9AG, United Kingdom. j.mullins@ed.ac.uk
SO Pflugers Archiv European Journal of Physiology, (1 Jan 2003) 445/4 (491-494).
Refs: 22
ISSN: 0031-6768 CODEN: PFLABK
CY Germany
DT Journal; Conference Article
FS 005 General Pathology and Pathological Anatomy
022 Human Genetics
LA English

L9 ANSWER 7 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:224267 CAPLUS
DN 138:380263
TI The Absence of SIR2.alpha. Protein Has No Effect on Global Gene Silencing in Mouse Embryonic Stem Cells
AU McBurney, Michael W.; Yang, Xiaofeng; Jardine, Karen; Bieman, Melissa; Th'ng, John; Lemieux, Madeleine
CS Ottawa Regional Cancer Centre and University of Ottawa, Ottawa, Can.
SO Molecular Cancer Research (2003), 1(5), 402-409
CODEN: MCROC5; ISSN: 1541-7786
PB American Association for Cancer Research
DT Journal
LA English
AB The ***yeast*** sir2 gene plays a central role in mediating gene silencing and DNA repair in this organism. The mouse sir2.alpha. gene is closely related to its ***yeast*** homolog and encodes a nuclear protein expressed at particularly high levels in embryonic stem (ES) cells. We used ***homologous*** ***recombination*** to create ES cells null for sir2.alpha. and found that these cells did not have elevated levels of acetylated histones and did not ectopically express silent genes. Unlike ***yeast*** sir2 mutants, our sir2.alpha. null ES cells had normal sensitivity to insults such as ionizing radiation and heat shock, and they were able to silence invading retroviruses normally. These sir2.alpha. null cells were able to differentiate in culture normally. Our results failed to provide evidence that the mammalian SIR2.alpha. protein plays a role in gene silencing and suggest that the physiol. substrate(s) for the SIR2.alpha. deacetylase may be nuclear proteins other than histones.
RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:142868 CAPLUS
DN 136:178954
TI Enhanced ***homologous*** ***recombination*** mediated by phage .lambda. recombination proteins
IN Court, Donald L.; Yu, Daiguan; Lee, E-Chiang; Ellis, Hilary M.; Jenkins, Nancy A.; Copeland, Neal G.
PA The Government of the United States of America, as Represented by the Secretary, Department of Health and Human Services, the National Institutes of Health and Human Services, USA
SO PCT Int. Appl., 124 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002014495	A2	20020221	WO 2001-US25507	20010814
WO 2002014495	A3	20020801		
WO 2002014495	B1	20021010		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2001083377	A5	20020225	AU 2001-83377	20010814
EP 1311661	A2	20030521	EP 2001-962178	20010814

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

US 2003224521	A1	20031204	US 2003-366044	20030212
US 2004092016	A1	20040513	US 2003-692553	20031023
PRAI US 2000-225164P	P	20000814		
US 2001-271632P	P	20010226		
WO 2001-US25507	W	20010814		
US 2003-366044	A1	20030212		

AB Disclosed herein are methods for generating recombinant DNA mols. in cells using ***homologous*** ***recombination*** mediated by recombinases and similar proteins. Such recombinases include the .lambda. proteins Beta, Exo, and Gam. The phage .lambda. recombinases are operably linked to a de-repressible promoter such as the .lambda. PL promoter, which is activated by temp. shift, thereby leading to expression of the .lambda. recombinases. Methods are also provided by inducing ***homologous*** ***recombination*** using single-stranded DNA mols., by introducing into the cell DNA capable of undergoing ***homologous*** ***recombination*** , and a single-stranded DNA-binding protein capable of promoting ***homologous*** ***recombination*** . Such single-stranded DNA binding proteins include .lambda. Beta, RecT, P22 Erf, and Rad52. The methods promote high efficiency ***homologous*** ***recombination*** in bacterial cells, and in eukaryotic cells such as mammalian cells. The methods are useful for cloning, the generation of transgenic and ***knockout*** animals, and gene replacement. The methods are also useful for subcloning large DNA fragments without the need for restriction enzymes. The methods are also useful for repairing single or multiple base mutations to wild type or creating specific mutations in the genome. Also disclosed are bacterial strains which are useful for high-efficiency ***homologous*** ***recombination*** . Thus, a highly efficient recombination for manipulating BAC DNA in Escherichia coli is described which uses a defective .lambda. prophage to supply functions that protect and recombine the electroporated linear DNA ***targeting*** cassette with the BAC sequence.

L9 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:755065 CAPLUS
DN 137:274035
TI Genome-wide deletion strategy, fungal ***target*** genes, methods to identify function of those genes, and uses for fungicide screening
IN Wang, Xun; Turgeon, Barbara Gillian; Yoder, Olen; Wu, Jianguo
PA USA
SO U.S. Pat. Appl. Publ., 65 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002142324	A1	20021003	US 2001-961527	20010924
PRAI US 2000-234650P	P	20000922		
US 2000-234673P	P	20000922		

AB A method is provided for gene identification and functional anal. using a genome-wide deletion strategy a genome-wide insertion strategy. The method may be used with any organism capable of ***homologous*** ***recombination*** , including plants, plant pathogens, microorganisms, and vertebrates. For example, a library of genomic DNA or cDNA inserts (DNA fragments) in a vector is contacted with an agent, e.g., a site specific endonuclease, which causes at least one double strand break in the DNA. The resulting gene ***knockout*** or gene insertion can then be screened for a desired phenotype. Satn. mutagenesis of the Cochliobolus heterostrophus genome was accomplished by random deletion of 8-10 kb fragments. The nucleotide sequences derived from Cochliobolus that code for polypeptides essential for normal fungal growth and development and/or for pathogenicity, and methods to identify polypeptides essential to the viability of an organism and/or those assocd. with pathogenicity are provided. The invention also includes methods of using these polypeptides to identify fungicides. The invention can further be used in a screening assay to identify inhibitors that are potential fungicides.

L9 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:309810 CAPLUS
DN 136:320318
TI Method for screening of DNA libraries and generation of recombinant DNA constructs utilizing .lambda. phage recombination function
IN Elledge, Stephen J.; Zhang, Pumin; Li, Mamie
PA Baylor College of Medicine, USA
SO U.S., 22 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6376192	B1	20020423	US 2000-724934	20001128
WO 2002044415	A1	20020606	WO 2001-US44088	20011127

W: AU, CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,

PT, SE, TR
AU 2002019856 A5 20020611 AU 2002-19856 20011127
PRAI US 2000-724934 A 20001128
WO 2001-US44088 W 20011127
AB The present invention provides methods of DNA library screening includes ***homologous*** ***recombination*** in E. coli utilizing lambda phage recombination functions. The advantage of the invention is to identify and select a gene of interest based on only about 60-100 bases of homol. from a DNA library and modify that gene fragment for use as a ***knockout*** ***targeting*** vector at the same time. Specifically, the method comprises inserting a pos. selection marker such as antibiotic resistance into the ***target*** sequence by ***homologous*** ***recombination*** facilitates isolation of ***target*** sequences and requires only about 58-100 base pairs of total homol., thus allowing the use of synthetic fragments of DNA for ***targeting*** . Once the clones are selected and cloned, they can then be sequenced and used to construct complete genes or cDNA sequences. DNA vector is designed for genomic library construction that features a novel genetic selection for inserts, automatic subcloning of isolated genomic clones and the presence of a neg. selection marker adjacent to the genomic inserts to facilitate later gene ***targeting*** .
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 11 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:169183 CAPLUS
DN 136:211850
TI Construction of gene ***knockout*** vectors using RKO clone and ***yeast*** ***targeting*** cassette ***homologous*** ***recombination*** in ***yeast***
IN Fisher, Katherine Elizabeth; Reaume, Andrew Gerard
PA Pfizer Products Inc., USA
SO Eur. Pat. Appl., 20 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1184461	A2	20020306	EP 2001-307021	20010817
EP 1184461	A3	20020522		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002291471	A2	20021008	JP 2001-250528	20010821

PRAI US 2000-228467P P 20000825
AB The present invention discloses methods of developing gene-***targeting*** vectors including transforming ***yeast*** cells with a RKO clone and a ***yeast*** ***targeting*** cassette, ***homologous*** ***recombination*** in ***yeast*** , and a subsequent seletion in E. coli, as well as uses of the vectors for directed mutation in a ***target*** gene, preferred in embryonic stem cells or whole animals. In particular, the invention discloses that the RKO clone comprises a genomic clone insert, a ***yeast*** replication element, a ***yeast*** selectable marker, a bacterial origin of replication, and a bacterial mammalian pos. selection marker, where the YTC comprises a bacterial/mammalian pos. seletion marker flanked by recombinogenic arms. The invention also provides compns. and methods for prepg. gene ***targeted*** mammalian cells and gene ***knockout*** mice.

L9 ANSWER 12 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:800 CAPLUS
DN 138:83907
TI Application of ***yeast*** genetics to biotechnology for producing anti-salinity plant
AU Yoshida, Kazuya
CS Graduate School of Bioscience, Nara Institute of Sciences and Technology, Ikoma-shi, Nara, 630-0101, Japan
SO Seibutsu Kogaku Kaishi (2002), 80(10), 482-485
CODEN: SEKAEA; ISSN: 0919-3758
PB Nippon Seibutsu Kogakkai
DT Journal; General Review
LA Japanese
AB A review. The technol. of genetic induction of useful ***yeast*** functional genes in plants by using recombination system with MAT (multiauto-transformation) vector was outlined. Prodn. of anti-salinity plant by introducing ***yeast*** genes such as the ENA1 gene for Na-ATPase involved in osmolality regulation was discussed. Some useful osmolality-regulating genes such as HKT1 and HKT2 for K+-Na+ cotransporter were isolated from rice. However, demonstration of physiol. activity of these genes is very difficult since prodn. of gene ***knockout*** plant models is difficult for the low ***homologous*** ***recombination*** activity in plants. The use of ***yeast*** gene ***knockout*** system for the screening of plant gene function was described to overcome this problem.

L9 ANSWER 13 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN DUPLICATE 2
AN 2002:571952 BIOSIS
DN PREV200200571952
TI Gene ***targeting*** by ***homologous*** ***recombination*** : A powerful addition to the genetic arsenal for Drosophila geneticists.
AU Rong, Yikang S. [Reprint author]
CS Laboratory of Molecular Cell Biology, National Cancer Institute, NIH, 37 Convent Dr., Bethesda, MD, 20892, USA
rongy@mail.nih.gov
SO Biochemical and Biophysical Research Communications, (September 13, 2002)
Vol. 297, No. 1, pp. 1-5. print.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 7 Nov 2002
Last Updated on STN: 7 Nov 2002
AB A series of recent publications have firmly established the notion that Drosophila researchers now have a general method to subject genes for ***targeted*** modification by ***homologous*** ***recombination*** (HR) (Science 288 (2000) 2013; Genetics 157 (3) (2001) 1307; Genes Dev. 16 (12) (2002) 1568; Genetics 161 (2002) 1125-1136). This method allows one to ***knockout*** essentially any gene starting with the DNA sequence of the gene. It has greatly enhanced studies of gene function as demonstrated by over 20 years of gene ***targeting*** practice in ***yeast*** and mouse. Here, I discuss the basic ***targeting*** methodology for eukaryotic organisms. I compare the Drosophila method with the traditional ***targeting*** scheme in ***yeast*** and mouse mainly to show that the ***targeting*** mechanism as well as many aspects of the experimental design remain unchanged, and that the Drosophila scheme differs only in the way in which the donor molecule for ***targeting*** is generated. I propose that the Drosophila method can be readily adapted in other organisms without culturable stem cells, since the mechanism for in vivo donor generation in Drosophila is likely to be functional in a variety of different organisms.

L9 ANSWER 14 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN
AN 2003:336821 BIOSIS
DN PREV200300336821
TI RIG-K, an Actin Cytoskeleton-Related Protein Is Involved in Cell Diferentiation Induced by ATRA.
AU Wang, Zhu-Gang [Reprint Author]; Luo, Hui-Jun [Reprint Author]; Huang, Qiu-Hua [Reprint Author]; Zhao, Qian [Reprint Author]; Sun, Xia [Reprint Author]; Wang, Long [Reprint Author]; Xu, Guo-Jiang [Reprint Author]; Yang, Hua [Reprint Author]; Chen, Sai-Juan [Reprint Author]; Chen, Zhu [Reprint Author]
CS State Key Lab of Medical Genomics, Shanghai Institute of Hematology, Rui-Jin Hospital, Shanghai, China
SO Blood, (November 16 2002) Vol. 100, No. 11, pp. Abstract No. 2124. print.
Meeting Info.: 44th Annual Meeting of the American Society of Hematology. Philadelphia, PA, USA. December 06-10, 2002. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 23 Jul 2003
Last Updated on STN: 23 Jul 2003
AB All trans retinoic acid (ATRA) can induce differentiation of acute promyelocytic leukemia (APL) cell strain NB4. It is known that ATRA functions through binding to retinoic acid receptors and regulates the expression of a series of ***target*** genes. In the process that ATRA induces differentiation of NB4 cells, not only nuclear factors, but also many membrane proteins are up-regulated. When screening the differentially expressed genes induced by ATRA, we cloned a new gene which is up regulated in NB4 cells when treated with ATRA. The sequence of this novel gene is highly homologous to mouse gene paladin which has been shown to colocalize with alpha-actinin in the stress fibers, focal adhesions, cell-cell junctions, and embryonic Z-lines. We named this new gene as Rig-K (retinoic acid induced gene-K). By using ***yeast*** two hybrid system we also found that Rig-K protein can interact with actin cytoskeleton related protein alpha-actinin, ABP280, Del-GEF etc. When Rig-K expression was blocked by riboenzyme specific to Rig-K in vitro, NB4 cell differentiation induced by ATRA is partially inhibited. It is suggested that Rig-K might play a key role in cell differentiation. To investigate the potential roles of Rig-K in cell growth, diffrentiation, especially, in hematopoiesis, we decided to create Rig-K knock out mouse model. We disrupted Rig-K gene through ***homologous*** ***recombination*** and got 7 recombined ES cell clones. As we expected, when wild type and ***targeted*** ES cells were induced to differentiate toward neuron cells, we found the ability of neurite outgrowth was severely impaired in Rig-K ***targeted*** ES cells. In Hela cells, Rig-K expression is dramatically down regulated when treated with PMA, following cell growth inhibition. The underlying mechanisms are being dissected. The results of phenotype analysis of Rig-K ***knockout*** mice will be presented.

L9 ANSWER 15 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:780937 CAPLUS
DN 135:340219
TI Bacterial multidrug resistance (MDR) efflux pumps and their uses
IN Davis, Deborah Vanriet; Rogers, Bruce Lee; White, Abigail Coffin

PA Phytera, Inc., USA
SO PCT Int. Appl., 139 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001079257	A2	20011025	WO 2001-US12230	20010412
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001053508	A5	20011030	AU 2001-53508	20010412

PRAI US 2000-197349P P 20000414
WO 2001-US12230 W 20010412

AB The invention features methods of detg. whether a nucleotide sequence encodes an multidrug resistance (MDR) efflux pump, methods for deleting a desired region of DNA in a bacterial cell, methods for detg. whether a test substance inhibits the growth or metab. of cells of a strain of E. faecalis bacteria having a disruptive mutation in a gene encoding a MDR efflux pump, methods for detg. whether a test substance includes a compd. that blocks efflux of an antibacterial agent from a cell, and methods for identifying an inhibitor of an MDR pump. Polynucleotide and polypeptide sequences for putative Enterococcus faecalis drug efflux proteins were identified by searching a genomic database for sequence homologs of several known MDR efflux pumps. E. faecalis was transformed with a pBS/Kan vector contg. flanking sequences for the ***target*** MDR gene and a selectable marker. The vector integrates into chromosomal DNA by ***homologous*** ***recombination*** . A second ***homologous*** ***recombination*** event results in a ***knockout*** strain which has deleted the selectable marker and the ***target*** gene. Multiple gene knockouts in a single strain can be generated using this method. As examples, growth of an E. faecalis .DELTA.NorA ***knockout*** strain was more sensitive to norfloxacin, a .DELTA.Abc7 ***knockout*** strain was more sensitive to daunorubicin and doxorubicin, and a .DELTA.Abc23 strain was more sensitive to clindamycin and lincomycin. The MDR gene ***knockout*** strains may be used to identify novel antibacterial agents or inhibitors of drug efflux pumps. The nucleotide sequences and polypeptides of this invention can also be used in drug screening assays in non-bacterial cells or cell-free systems.

L9 ANSWER 16 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 3
AN 2002:171865 BIOSIS
DN PREV200200171865
TI Cloning of the PpMSH-2 cDNA of Physcomitrella patens, a moss in which gene ***targeting*** by ***homologous*** ***recombination*** occurs at high frequency.
AU Brun, Florent; Gonneau, Martine; Doutriaux, Marie-Pascale; Laloue, Michel; Nogue, Fabien [Reprint author]
CS Laboratoire de Biologie Cellulaire, INRA, Route de St.-Cyr, 78026, Versailles, France
nogue@versailles.inra.fr
SO Biochimie (Paris), (November-December, 2001) Vol. 83, No. 11-12, pp. 1003-1008. print.
CODEN: BICMBE. ISSN: 0300-9084.
DT Article
LA English
ED Entered STN: 5 Mar 2002
Last Updated on STN: 5 Mar 2002
AB In the moss Physcomitrella patens integrative transformants from ***homologous*** ***recombination*** are obtained at an efficiency comparable to that found for ***yeast*** . This property, unique in the plant kingdom, allows the ***knockout*** of specific genes. It also makes the moss a convenient model to study the regulation of ***homologous*** ***recombination*** in plants. We used degenerate oligonucleotides designed from AtMSH2 from Arabidopsis thaliana and other known MutS homologues to isolate the P. patens MSH2 (PpMSH2) cDNA. The deduced sequence of the PpMSH2 protein is respectively 60.8% and 59.6% identical to the maize and A. thaliana MSH2. Phylogenic studies show that PpMSH2 is closely related to the group of plant MSH2 proteins. Southern analysis reveals that the gene exists as a single copy in the P. patens genome.

L9 ANSWER 17 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:829999 CAPLUS
DN 133:359775
TI Method for constructing gene- ***targeting*** vectors, transgenic organism preparation, and cDNA library screening
IN Akiyama, Kiyotaka; Sasai, Taira; Watabe, Hirotaka
PA Japan Tobacco, Inc., Japan
SO Jpn. Kokai Tokkyo Koho, 59 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2000325091	A2	20001128	JP 2000-81795	20000317

PRAI JP 1999-71390 A 19990317

AB A method for constructing vectors used for gene ***knockout*** via ***homologous*** ***recombination*** is disclosed. Use of gene markers such as neomycin resistance (neo) gene, reporter genes such as luciferase (luc) gene or .beta.-lactamase gene, BAC, YAC, and genomic DNA library, are claimed. A method for constructing transgenic cell, bacteria, virus, or mammals from embryonic stem cells using the vector is also claimed. A method for screening cDNA libraries is also claimed. We developed a simple and rapid method for constructing gene ***knockout*** vectors using inverse-PCR (IPCR). The method consists of three steps: (i) digestion of a ***target*** bacterial artificial chromosome with several restriction enzymes (six-base cutters) followed by self-ligation; (ii) IPCR using circular DNAs as templates and two primers which are oriented in opposite directions; and (iii) cloning into a vector contg. a pos. selection marker, which results in a typical replacement ***knockout*** vector. We successfully ***targeted*** three mouse genes including the HPRT gene using this method. Compared with the conventional method, this method requires much less time (no more than 3 wk). Notably, this method requires only small amts. of sequence information (several hundred base pairs such as is available from expressed sequence tags) and can be extended to a systematic mass prodn. of ***targeting*** vectors applicable to many organisms, including ***yeast*** .

L9 ANSWER 18 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:311319 CAPLUS
DN 130:333733
TI Efficient construction of gene ***targeting*** vectors and use in generation of ***knockout*** mice
IN Woychik, Richard; Garfinkel, David
PA Amgen Inc., USA
SO PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9923239	A1	19990514	WO 1998-US11388	19980603
W: AU, CA, JP, MX				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6090554	A	20000718	US 1997-963602	19971031
CA 2308016	AA	19990514	CA 1998-2308016	19980603
AU 9877213	A1	19990524	AU 1998-77213	19980603
EP 1025252	A1	20000809	EP 1998-925209	19980603
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001521749	T2	20011113	JP 2000-519094	19980603

PRAI US 1997-963602 A 19971031
WO 1998-US11388 W 19980603

AB The present invention is directed to highly efficient methods for prepg. gene ***targeting*** vector by exploiting certain cells to mediate ***homologous*** ***recombination*** . The generation of ***targeting*** vectors in ***yeast*** strain DG1500 by ***homologous*** ***recombination*** was exemplified by the use of the Tg737 gene. A specific engineered fragment (SEF) contg. a market cassette being flanked on each side by mammalian gene-specific flaking sequences homologous to a portion of the gene to be ***targeted*** was generated and then recombined with a shuttle vector. The ***targeted*** mutations were generated in embryotic stem (ES) cells using the above vector and cells from one of the ES clones were used for injections into C57/BL/6 blastocysts to generate Tg737 ***knockout*** mice. Different methods for producing or obtaining gene ***targeting*** constructs by way of ***homologous*** ***recombination*** in host cells and to ***targeting*** constructs produced by those methods were also described.

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 19 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 4
AN 1999:345127 BIOSIS
DN PREV199900345127
TI Stimulation of ***homologous*** ***recombination*** in plants by expression of the bacterial resolvase RuvC.
AU Shalev, Gil; Sitrit, Yaron; Avivi-Ragolski, Naomi; Lichtenstein, Conrad; Levy, Avraham A. [Reprint author]
CS Department of Plant Sciences, Weizmann Institute of Science, Rehovot, 76100, Israel
SO Proceedings of the National Academy of Sciences of the United States of America, (June 22, 1999) Vol. 96, No. 13, pp. 7398-7402. print.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 24 Aug 1999
Last Updated on STN: 24 Aug 1999
AB ***Targeted*** gene disruption exploits ***homologous***

recombination (HR) as a powerful reverse genetic tool, for example, in bacteria, ***yeast*** , and transgenic ***knockout*** mice, but it has not been applied to plants, owing to the low frequency of HR and the lack of recombinogenic mutants. To increase the frequency of HR in plants, we constructed transgenic tobacco lines carrying the Escherichia coli RuvC gene fused to a plant viral nuclear localization signal. We show that RuvC, encoding an endonuclease that binds to and resolves recombination intermediates (Holliday junctions) is properly transcribed in these lines and stimulates HR. We observed a 12-fold stimulation of somatic crossover between genomic sequences, a 11-fold stimulation of intrachromosomal recombination, and a 56-fold increase for the frequency of extrachromosomal recombination between plasmids cotransformed into young leaves via particle bombardment. This stimulating effect may be transferred to any plant species to obtain recombinogenic plants and thus constitutes an important step toward gene ***targeting*** .

L9 ANSWER 20 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN DUPLICATE 5
AN 1999:317264 BIOSIS
DN PREV199900317264
TI Construction of gene ***targeting*** vectors from lambdaKOS genomic libraries.
AU Wattler, Sigrid; Kelly, Mike; Nehls, Michael [Reprint author]
CS Lexicon Genetics Incorporated, 4000 Research Forest Drive, Woodlands, TX, 77381, USA
SO Biotechniques, (June, 1999) Vol. 26, No. 6, pp. 1150-1160. print.
CODEN: BTNQDO. ISSN: 0736-6205.
DT Article
LA English
ED Entered STN: 17 Aug 1999
Last Updated on STN: 17 Aug 1999
AB We describe a highly redundant murine genomic library in a new lambda phage, lambda ***knockout*** shuttle (lambdaKOS) that facilitates the very rapid construction of replacement-type gene ***targeting*** vectors. The library consists of 94 individually amplified subpools, each containing an average of 40000 independent genomic clones. The subpools are arrayed into a 96-well format that allows a PCR-based efficient recovery of independent genomic clones. The lambdaKOS vector backbone permits the CRE-mediated conversion into high-copy number pKOS plasmids, wherein the genomic inserts are automatically flanked by negative-selection cassettes. The lambdaKOS vector system exploits the ***yeast*** ***homologous*** ***recombination*** machinery to simplify the construction of replacement-type gene ***targeting*** vectors independent of restriction sites within the genomic insert. We outline procedures that allow the generation of simple and more sophisticated conditional gene ***targeting*** vectors within 3-4 weeks, beginning with the screening of thelambdaKOS genomic library.

L9 ANSWER 21 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on
STN
AN 1997:391337 BIOSIS
DN PREV199799690540
TI Transposon-generated 'knock-out' and 'knock-in' gene- ***targeting*** constructs for use in mice.
AU Westphal, Christoph Heiner [Reprint author]; Leder, Philip
CS Dep. Genetics, Harv. Med. Sch., 200 Longwood Ave., Boston, MA 02115, USA
SO Current Biology, (1997) Vol. 7, No. 7, pp. 530-533.
CODEN: CUBLE2. ISSN: 0960-9822.
DT Article
LA English
ED Entered STN: 10 Sep 1997
Last Updated on STN: 10 Sep 1997
AB The conventional technique for ***targeted*** mutation of mouse genes entails placing a genomic DNA fragment containing the gene of interest into a vector for fine mapping, followed by cloning of two genomic arms around a selectable neomycin-resistance cassette in a vector containing thymidine kinase (1); this generally requires 1-2 months of work for each construct. The single 'knock-out' construct is then transfected into mouse embryonic stem (ES) cells, which are subsequently subjected to positive selection (using G418 to select for neomycin-resistance) and negative selection (using FIAU to exclude cells lacking thymidine kinase), allowing the selection of cells which have undergone ***homologous*** ***recombination*** with the ***knockout*** vector. This approach leads to inactivation of the gene of interest (2). Recently, an in vitro reaction was developed, on the basis of the ***yeast*** Ty transposon, as a useful technique in shotgun sequencing (3). An artificial transposable element, integrase enzyme and the ***target*** plasmid are incubated together to engender transposition. The DNA is then purified, and subsequently electroporated into bacteria. The transposon and the ***target*** plasmid bear distinct antibiotic resistance markers (trimethoprim and ampicillin, respectively), allowing double selection for transposition events. In the present study, we have modified this system to allow the rapid, simultaneous generation of a palette of potential gene ***targeting*** constructs. Our approach led from genomic clone to completed construct ready for transfection in a matter of days. The results presented here indicate that this technique should also be applicable to the generation of gene fusion constructs (4-8), simplifying this technically demanding method.

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